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Studies on embryonic lethal characters in the domestic fowl

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STUDIES ON EMBRYONIC LETHAL CHARACTERS IN THE DOMESTIC FOWL

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ABSTRACT

The studies reported involved the examination of 4,180 dead embryos of known pedigree from 16,462 fertile eggs produced by 567 hens mated to 86 sires and involving several types of special matings. Particular consideration was given to the age of the embryos at death, to distribution of the sexes and to descriptions of all types of abnormal embryos encountered; as influenced by time of hatch, by breed, by character of mating, by sire and by dam. Tests were made of the possible inheritance of commonly encountered anomalies. Mortality curves were of the same general type as reported by previous investigators but varied appreciably at certain periods of incubation for different breeding stock. Slightly more than 60 per cent of the dead embryos (when all dead embryos were examined) or over fifteen per cent of the fertile eggs set, were classified as being abnormal in some way. Abnormalities of the eyes increased markedly in incidence as the season advanced, while the incidence of hemorrhage decreased for later hatches. Season had little affect, however, upon the occurrence of most types of abnormal embryos. Limited breeding data indicated that chondrodystrophy is inherited, and that if so the mode of inheritance is complex. The hypothesis is suggested that no innate differences underlie specific malpositions but rather that an impaired sense of orientation is perhaps the primary consideration. Evidence presented supports the view that breeding is an important factor in the incidence of malpositions. It is believed that a sex-linked lethal gene was responsible for aberrant sex ratios observed in certain instances. Dwarfism is shown to be inherited as a simple autosomal recessive. Breeding results indicate that (a) dwarfism and stickiness are controlled by separate genes, (b) creeper and dwarfism are distinct traits and (c) chondrodystrophy is not associated in inheritance with any of these characters.

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STUDIES ON EMBRYONIC LETHAL CHARACTERS IN THE DOMESTIC FOWL*

INTRODUCTION

The problem of reproducing the flock with greatest efficiency is of primary importance to all poultrymen. Under existing conditions, it is necessary to set an average of fifty per cent more eggs than the number of chicks expected. Furthermore, it is necessary to start in the brooders three to four chicks for every mature pullet desired. This inefficiency is due largely to mortality of the embryos and of the subsequent chicks. It seems plausible to believe that some of this mortality may be caused by inherited lethal genes. The present study was undertaken in an attempt to ascertain the existence of such genes in breeding stock of the more popular breeds and to determine the mode of inheritance of lethals encountered.

Advancement has been made in the past decade toward the perfection of mechanical equipment used in incubating the eggs and in brooding the chicks. Tremendous strides have been taken toward the better nutrition of growing chicks and of breeding birds so that losses resulting from physiological disturbances have been greatly reduced. The control of disease has likewise advanced rapidly. But improvement in reproductive efficiency by the elimination of inherited defects from the breeding stock has received scant attention.

Extensive observations have been made in the field of abnormal embryology of the chick, but little has been done to determine the possible inheritance of abnormalities observed. There is little question that abnormalities frequently arise from other than inherent causes, but until extensive tests are made to ascertain whether or not a given abnormality is inherited, the assumption of non-inheritance remains a mere guess. When it is estabished that a given lethal character is inherited steps may be taken to eliminate it from the flock.

The great number of inherited lethal genes which have been discovered in Drosophila and the identification of a number of inherited lethals in larger animals points to the probability that a number of them exist in the domestic fowl.

* The work presented in this bulletin was conducted at Iowa State College and at Louisiana State University. It was accepted at Iowa State College as a dissertation in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

REVIEW OF LITERATURE

Henderson (1930) gives an adequate classification of the most important known influences bearing upon embronic life and presents a comprehensive review of the literature covering all phases of the problem.

Hatchability of eggs or its component factors has been shown to be inherited. Pearl and Surface (1909) and Pearl (1911) indicated that differences in hatchability were probably inherited. Hays and Sanborn (1924) concluded that high hatchability is dependent upon one dominant gene. Dunn (1922 and 1923a) however issued the warning that, "The first advance we can make over this (i. e. considering hatchability as a general trait) is to abandon the concept of hatchability as a single or simple character . . . . the logical method of approach is to study separate causes singly wherever that is possible." Hays (1926) has offered evidence that the male is an important factor in the determination and inheritance of hatchability. Jull (1928) states, "All of the available evidence to date concerning factors affecting hatchability points to the conclusion that hatchability is inherited . . . . Undoubtedly malformations are responsible for a goodly proportion of fully formed chicks that fail to hatch and this aspect of the subject should be investigated much more thoroughly than has been done up to the present." Snyder (1931) reports that, ". . . . somewhat encouraging results are being obtained which would suggest the possibility of establishing families more or less homozygous for high hatching ability, and others for low hatching ability." Jull (1930) concludes, contrary to the hypothesis of Hays and Sanborn, that probably more than one pair of genes is involved in the inheritance of hatchability. Jull (1932) gives a comprehensive review of work bearing upon the inheritance of hatchability and concludes, "Undoubtedly several pairs of genes determine hatchability, and it is apparent that different pairs of genes vary in their effects . . . ."

The influence of malpositions of embryos upon hatching quality has been investigated rather extensively. Sanctuary (1925), Hutt (1929), Smith (1931), Byerly and Olsen (1931) and (1932), Taylor (1932) and Hutt and Pilkey (1934) have all published work bearing upon some phase of this problem. The three first named investigators have expressed the belief that malpositions may be inherited, but conclusive evidence has not been submitted. Kuo (1932) discusses at some length seven different critical stages in the normal development of the avian embryo and the mechanics involved in the emergence of the chick from the shell. He states that malpositions result if the embryo fails to pass successfully any one of the seven critical stages.

Extensive descriptions, illustrations, and classifications of teratological monsters in chick embryos have been made by Dareste (1891), Stockard (1921), Lesbre (1927), Hutt and Greenwood (1929b), Hutt (1930a) and Hutt and Pilkey (1930), but conclusive evidence as to the inheritance of different types of monsters is lacking.

Payne (1919), Byerly (1930), Riddle (1930), Smith (1933) and Byerly, Knox, and Jull (1934) have published results concerning the distribution of mortality during the period of incubation and have offered some explanations as to the causes of the two sharply defined peaks or modes of mortality which occur, one near the beginning of embryonic development, the other near the end of the incubation period.

Wriedt (1930) discusses at length the lethal factors known in domestic animals. Concerning the occurrence of lethals in livestock, this author states, "Among cattle seven recessive lethal factors have already been noted, among horses one, among swine one, and among sheep two, and this in spite of the fact that investigations of lethal factors among livestock are not general."

Three definite embryonic lethal factors have been reported in domestic fowls. Dunn (1923a) reported a recessive embryonic lethal factor linked with recessive white plumage color in Wyandotte stock. This factor appeared to exert its influence relatively early in the incubation period.

Dunn and Landauer (1926) and Landauer and Dunn (1930a) and Landauer (1930) and (1932) have offered conclusive proof that in the homozygous condition the creeper trait, which is determined by a single dominant gene, is lethal. The characteristic lethal period is designated as beginning on the fourth day of incubation although a small percentage of homozygous embryos survive until the last week of incubation and in rare instances are alive at hatching time, but they never hatch.

An inherited recessive character designated as "sticky" has been investigated by Byerly and Jull (1930) and (1932) and Byerly (1931), which is lethal in the homozygous condition. The outstanding characteristics of such embryos are the extreme softness of the bones and the unabsorbed amniotic and allantoic fluids which are very viscous at hatching time. A general edema is usually noted. The lethal action occurs during the latter part of the incubation period and, as stated by Byerly and Jull (loc. lit.), "Stickiness . . . . permits life withing the shell, but is a bar to hatching."

Crew (1925) reported the dominant factor for frizzled plumage as being lethal when homozygous but later work by Hutt (1930b), Landauer and Dunn (1930b) and Landauer (1930) failed to reveal either a gametic or a zygotic lethal factor associated with this

character although excessive embryonic mortality occurred in frizzled stock. This was explained as being due to the action of physiological factors.

Chondrodystrophy in the fowl was first reported by Landauer and Dunn (1926). Considerable work has been done since its discovery to determine whether or not chondrodystrophy is inherited. Dunn's (1927) results militate strongly against genetic recombinations as a causal factor, however he states, "the distribution of chondrodystrophy among the descendents of the inbred 'chondro' family, irregular and rare as it is, might still be reconciled with the theory of inheritance if the hereditary basis of chondrodystrophy were not a single factor but either a combination of several recessive factors or a general hereditary background determining susceptibility to external or internal conditions which disturb development." Hutt and Greenwood (1929a) found no semblance of genetic ratios and report negative evidence for chondrodystrophy being due to genetic constitution of the embryo but they state, "However, the greater frequency of the abnormality in the progeny of certain birds of our flock than from others suggests strongly that the tendency to produce it is inherited." Munro (1932) concludes, ". . . . the evidence is strong for believing the malformation to be due to genetic factors which depend on unfavorable environmental conditions to become phenotypically expressed." Byerly, Titus and Ellis (1933a and b) on the other hand, have concluded that chondrodystrophy is not an inherited character. It appears, then, although evidence offered to date is somewhat conflicting, that chondrodystrophy may be inherited but, if so, the mode of inheritance is complex.

Connecticut (Storrs) Agr. Expt. Sta. Rpt. (1926) states that ". . . . the pure rumpless condition is not necessarily lethal, since one rumpless fowl has been found to be pure, and to produce only rumpless chicks when bred to normal males."

Landauer (1929) reported a case of thyrogenous dwarfism in the Rhode Island Red fowl. Careful histological studies were made of several glands, but no data were available as to the inheritance of this condition. Mayhew and Upp (1932) and Upp (1932) have offered limited evidence which indicated that dwarfism is inherited as a recessive character and that it has a delayed lethal action. Death is occasioned apparently by a failure of certain ductless glands to function properly.

EXPERIMENTAL

Materials and Methods

The material used in this study was collected over a three-year period, 1931 to 1933 inclusive, and at two different institutions. The first work was done during the spring of 1931 at Iowa State College. Examination was made of 206 dead embryos from 653 fertile eggs set from 26 White Wyandotte females mated to four males. One hundred and forty-seven additional embryos, representing only a portion of those that died from 532 fertile eggs, were examined from 15 other dams of various breeds.

During the fall of 1931 and the spring of 1932, a total of 974 embryo examinations was made at Louisiana State University. This was the total number of dead embryos from 3,843 fertile eggs set from 159 dams mated to 28 sires. Of the individual matings, 31 were S. C. W. Leghorns, 104 Rhode Island Reds, 5 White Wyandottes and 19 cross-breds. At Iowa State College in the spring of 1932, a total of 473 embryos that died on or after the 14th day was examined from 89 closely inbred S. C. W. Leghorn hens mated to 8 males of like breeding. A total of 2,733 fertile eggs was set from these hens during the season of examination.

A total of 1,163 dead embryos was examined at Louisiana State University in 1933. These embryos were obtained from 4,082 fertile eggs produced by 133 hens mated to 25 sires. The individual matings were as follows: 56 S. C. W. Leghorn matings, of which about one-half were mildly inbred matings, 15 R. I. Red, 4 creeper, 6 White Wyandotte and 52 matings of cross-breds and R. I. Reds.

Also in 1933 at Iowa State College 1,217 dead embryos (14 days or older) were examined from 4,619 fertile eggs laid by 147 highly inbred S. C. W. Leghorn hens mated to 12 inbred Leghorn males.

This study involved, then, 4,180 dead embryos of known pedigree from 16,462 fertile eggs produced by 567 hens mated to 86 sires and involving several types of special matings.

Description of Abnormalities Encountered

Chondrodystrophy. This abnormality varies greatly in degree of expression, ranging from embryos mildly affected to extremely abnormal embryos. The most common characteristics of chondrodystrophic embryos are "parrot beak" resulting from shortened lower beak and downward curvature of upper beak; skull bulged anteriorly, causing head to have a globular contour; greatly shortened and thickened legs; retarded down growth; and a general retarded size.

Creeper. The distinguishing characteristic of heterozygous creeper embryos is the markedly shortened metatarsi. Homozygous creeper

embryos characteristically die on the third or fourth day of incubation but some embryos live up to near the end of the incubation period. They resemble extreme chondrodystrophic embryos, but the head changes are less pronounced. The eyelids are absent or greatly reduced in size. In the homozygous creeper embryo size is retarded; and all extremities are greatly shortened, the legs appear as little more than feet attached to the body, but the long bones are represented in vestigial condition.

Sticky. The embryo is immersed in a sticky, viscous liquid; shows retarded size; a general edema is typical; and the bones are very soft and rubbery.

Microphthalmia. The failure of one or both eyes to develop normally is designated as microphthalmia. The condition varies from a scarcely noticeable decrease in the size of the eye to an apparent absence of the eye. Hutt and Greenwood (1929a) consider anophthalmia as merely an advanced degree of microphthalmia.

Ectopia or eversion of viscera. Such embryos are characterized by failure of the body cavity to close which leaves the viscera exposed. The condition occurs in varying degrees.

Hemorrhage. The incidence of hemorrhage was sub-divided according to the location of the rupture, into the following classes: general internal hemorrhage in which the point of rupture is not detected; external hemorrhage at pipping time; and hemorrhage at heart, liver, kidneys or brain.

Edema. Classed as general; enlarged Musculus Complexus; and edema around head and throat. Infiltration with liquid of parts affected is the distinguishing characteristic.

Constricted Membranes. Such embryos were characterized by a constriction of the extra-embryonic membranes in such a manner as to interfere with normal development. Various parts of the body were affected in different embryos.

Retarded Size. Embryos were observed that showed developmental characteristics of a given age but in size of body were distinctly smaller as compared to normal embryos of like development. For example, an embryo might show normal development of a 16 day embryo in all respects except size, but in body size was comparable to a 13 day embryo.

Delayed Hatch. This term was used to designate embryos that were slow to hatch as compared to others of the same setting. For example, embryos were found that were still alive in the shell on the 24th day of incubation that had not developed past the normal 20th day stage. Such an embryo would be classified as "delayed" four days.

Yolk Excluded. This term designated those individuals in which the body cavity was practically closed with the yolk remaining outside the body.

Prognathia. Upper and lower beak of uneven length.

Hyperencephaly. Complete absence of upper beak, absence of roof of the cranium and absence of both eyes.

Exencephaly. Brain exposed.

Otocephaly. Used in this study to designate extreme otocephaly or complete absence of the head.

Duplicity. The one case of duplicity recorded was a case of practically complete twinning. The embryos are joined firmly in the abdominal region.

An Example of complete twinning was also found in which the twins were entirely separate.

Malpositions. The normal hatching position is described by Hutt (1929) as follows: "the head is toward the larger end of the egg. The neck is so bent as to bring the head to the right side of the body and backwards with the beak under the right wing and just external to femorotibial joint. As a consequence of this position the tip of the beak rests near the shell at a point just where the inner shell membrane separates from the outer to form the air-cell. The feet are folded on the ventral side of the body almost exactly the same as in a trussed fowl, except that the toes reach to the head."

Several major malpositions occur commonly. They were classified as follows for this study:

Malposition 1. Head between thighs. Sanctuary (1925).

Malposition 2. Head in the small end of egg. Reaumur (1751).

Malposition 3. Head to the left. Sanctuary (1925).

Malposition 4. Embryo rotated in the shell. Hutt (1929).

Malposition 5. Head not under the wing. Hutt and Cavers (1931).

Malposition 6. Head to the left and not under wing. Positions 3 and 5 combined.

Malposition 7. Embryo rotated and head not under wing. Positions 4 and 5 combined.

Malposition 8. Head between thighs and in small end of egg. Positions 1 and 2 combined.

Malposition 9. Feet over head. Smith (1931).

Dwarfism. This abnormality is in some cases noticeable in the older embryos or at hatching time but in others cannot be detected until the chicks are three to five weeks of age. Distinguishing characteristics are; shortness of legs, particularly the metatarsi; outer toes turn more or less outward and backward; body carriage is ab-

normal, the fore part of the body being carried lower than is normal; the head is shorter and broader, with a "parrot" beak; the eyes protrude and the skin around the head is wrinkled and thickened.

Routine Examination of the Embryos

The first year, the eggs were candled every other day from the 4th to the 18th day of incubation, and examinations made on the day of candling. The dead-in-shell were examined on the day that the chicks were pedigreed. For the examinations made at Louisiana State University the eggs were candled on the 8th or 9th day of incubation and again on the 17th or 18th day of incubation, and the dead embryos examined the same day as candled. The dead-in-shells and the embryos still alive were examined on the 22nd day of incubation, or later.

Prior to examination the eggs were separated by hens and arranged in order by pens. Record for each unhatched egg was made as to dam number, pen number, date eggs were set, date examination was made, age of embryo at death, sex of embryo and "notes." The description of all abnormalities observed was recorded under notes. The age of the embryo at death was judged by the relative development of the embryo and of the extra-embryonic membranes.

The process of examination was started by opening the egg rather carefully with forceps and a pair of small scissors. The shell was removed from the large end of the egg and the shell membranes carefully removed. The position in the shell of the more advanced embryos was recorded. The embryos were removed from the shell and apparent external abnormalities noted. They were dissected to observe internal abnormalities if present and to determine sex. To make the internal examination the embryo was held with the back uppermost and was cut completely across the back to a point about one-third the distance down each side. The incision was made in the middle region of the lungs, thus being somewhat anterior to the gonads. Incisions were then made parallel to the dorsal line of the body and extending caudally to the region of, but ventral to the hip joints. The anterior end of the excised portion of the back was then grasped with blunt forceps, gently raised and turned posteriorly exposing the gonads and kidneys. After the sex was determined, the viscera were examined and gross abnormalities, if any, noted. Brittleness of the bones was gauged by breaking the matatarus or the tibia of the right leg of the older embryos.

Special consideration has been given to the age of the embryos at death, distribution of the sexes and to descriptions of all types of abnormal embryos encountered; as influenced by time of hatch, by breed, by character of mating, by sire and by dam.

Matings were made in attempts to determine the possible inheritance of certain embryonic abnormalities, particularly chondro-

dystrophy, sex ratio and mal-positions. In determining the mode of inheritance of dwarfism and the inter-relationships of dwarfism, creeper and stickiness characters, matings were made to produce F_1, F_2, and backcross generations.

The eggs were incubated in cabinet-type, forced draft incubators under controlled conditions. Settings were made at Louisiana State University at all seasons of the year although most of the eggs were set during the spring, or normal hatching season. All eggs at Iowa State College were set between January 9 and May 6, with a large percentage set during the middle of the season.

All Louisiana breeding birds were allowed outside runs throughout the year with some green feed crop always available. The Iowa breeders were confined from January until April but were supplied with cod liver oil in the ration during the breeding season.

RESULTS

MORTALITY CURVES

Tables 1 and 2, figures I, II and III present complete embryo mortality for 9,110 fertile eggs and also give the mortality of the third week of incubation for 7,352 additional fertile eggs.

Character of Mating. Table 1 and figures I and II present the mortality distribution for groups separated according to the character of the matings. The types of matings used consisted of regular matings, i.e., those other than the various special matings given below; mildly inbred matings; matings in which only the sire was a carrier of dwarfism; matings in which both parents carried dwarfism; matings in which both sire and dam carried dwarfism and sire also carried stickiness; matings in which both parents carried stickiness; closely inbred S. C. W. Leghorns (was also malposition stock); matings in which both parents were creepers; matings in which both parents carried Kiwi or silky plumage; and matings in which the sire carried the albino character, Warren (1933a).

Although the mortality curves are similar for all except one of the various kinds of matings, some notable differences are evident. Mortality for the regular, the mildly inbred, the sire dwarf carrier and the kiwi matings is quite similar throughout and hatchability was appreciably higher for these groups than for others. Mortality for the first to the fourth day of incubation inclusive was appreciably higher for the stickiness, the albino and the creeper matings. Exceptionally high 18th day mortality is noted for the stickiness and the both parents dwarfism carrier sire also stickiness carrier matings, with dwarf matings intermediate between these and the higher hatching groups mentioned previously. The exceptionally high 18th day mortality is due to the lethal action of the stickiness gene at

TABLE 1

Daily Embryo Mortality

By Character of Mating

Character of mating[1]	Total No. dead embryos	Total fertile eggs set	Percent dead of fertile eggs set													
			Days of incubation													
			1	2	3	4	5	6	7	8	9	10	11	12	13	14
0	1458	5521	.72	1.56	2.72	1.25	1.10	.71	.62	.71	.49	.62	.67	.58	.25	.31
1	211	983	.41	.71	1.42	.51	.41	.31	.61	.31	.41	.31	.81	.51	.20	.10
2	2367	7352	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.10	.11	.14	.22
3	132	481	1.25	2.49	4.37	1.87	.83	2.08	.83	.42	.42	.21	0.00	.21	0.00	0.00
4	289	794	.50	2.90	3.90	1.89	1.13	.38	1.26	.50	.38	.38	0.00	.25	.13	.13
6	39	62	1.61	3.22	9.68	3.22	0.00	0.00	0.00	1.61	0.00	1.61	1.61	0.00	0.00	0.00
9	55	84	0.00	0.00	3.57	2.38	0.00	1.19	0.00	1.19	2.38	1.19	2.38	2.38	1.19	0.00
10	45	222	0.00	1.35	0.00	.90	.90	.45	.90	1.35	.90	0.00	0.00	.45	0.00	0.00
11	94	180	0.00	1.11	5.00	1.11	.56	0.00	1.11	.56	2.22	1.67	2.78	4.44	0.00	.56
15	115	225	0.00	.89	2.67	1.33	.44	.44	.44	.44	.89	.44	.44	.44	.44	.44

TABLE 1 (Continued)

Daily Embryo Mortality

By Character of Mating

Character of mating[1]	Total No. dead embryos	Total fertile eggs set	Percent dead of fertile eggs set												
			Days of incubation												
			15	16	17	18	19	20	21	22	23	24	25	Age at death unknown	Alive at 21st day
0	1458	5521	.72	.49	1.27	3.44	3.30	2.57	.96	.60	.24	.13	0.00	.24	1.63
1	211	983	.41	.31	.61	4.88	4.37	1.32	.51	.61	.51	.61	0.00	0.00	.31
2	2367	7352	.36	.26	.91	2.17	4.08	6.18	7.96	.....	.....	.....	.....	10.50	.....
3	132	481	.21	.21	1.04	5.61	2.29	1.16	0.00	0.00	.42	.21	0.00	.83	0.00
4	289	794	.13	.25	1.39	8.69	6.68	3.78	1.13	.13	.25	.25	0.00	0.00	0.00
6	39	62	1.61	0.00	1.61	24.19	6.45	8.84	0.00	0.00	1.61	0.00	0.00	0.00	0.00
9	55	84	0.00	3.57	7.14	11.90	8.33	11.90	2.38	0.00	1.19	1.19	0.00	0.00	0.00
10	45	222	.45	0.00	0.00	.90	4.95	4.05	1.35	0.00	.90	.90	0.00	0.00	0.00
11	94	180	2.22	.56	1.67	8.33	8.89	4.44	1.11	0.00	1.11	2.22	.56	0.00	0.00
15	115	225	2.22	2.67	6.67	12.89	8.89	5.33	1.78	0.00	.44	.44	0.00	0.00	0.00

[1] Legend for Character of Mating.

0. Regular.
1. Mildly inbred.
2. Highly inbred.
3. Sire dwarf carrier.
4. Both sire and dam dwarf carriers.
6. Both sire and dam sticky carriers.
9. Both parents creepers.
10. Kiwi mating.
11. Albino Wyandotte.
15. Sire sticky carrier; both parents dwarf carriers.

FIGURE I

EMBRYONIC MORTALIT'' - BY CHARACTER OF MATING

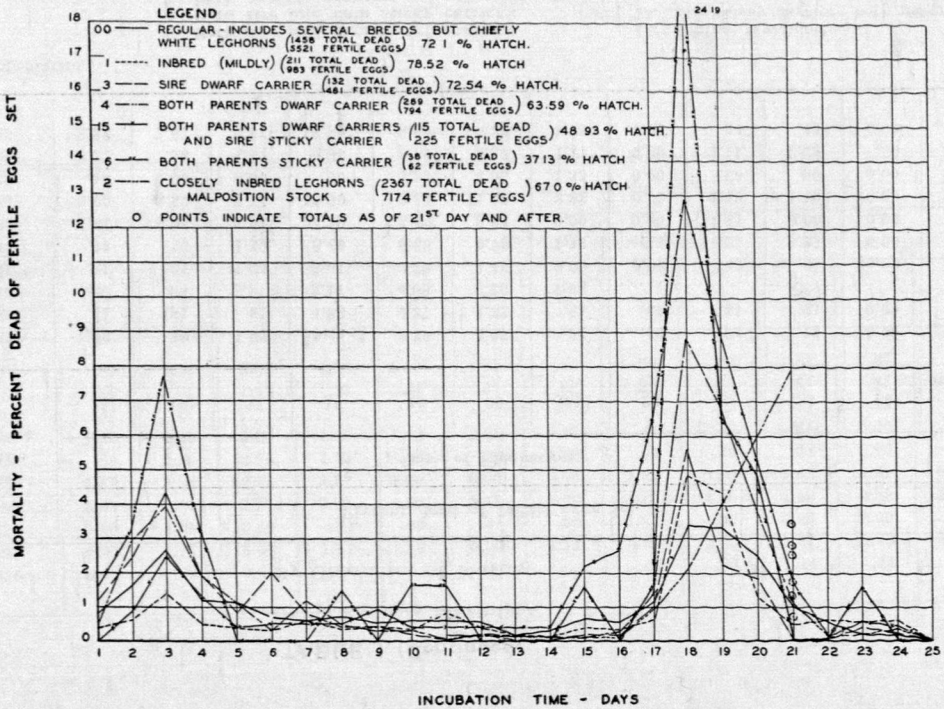
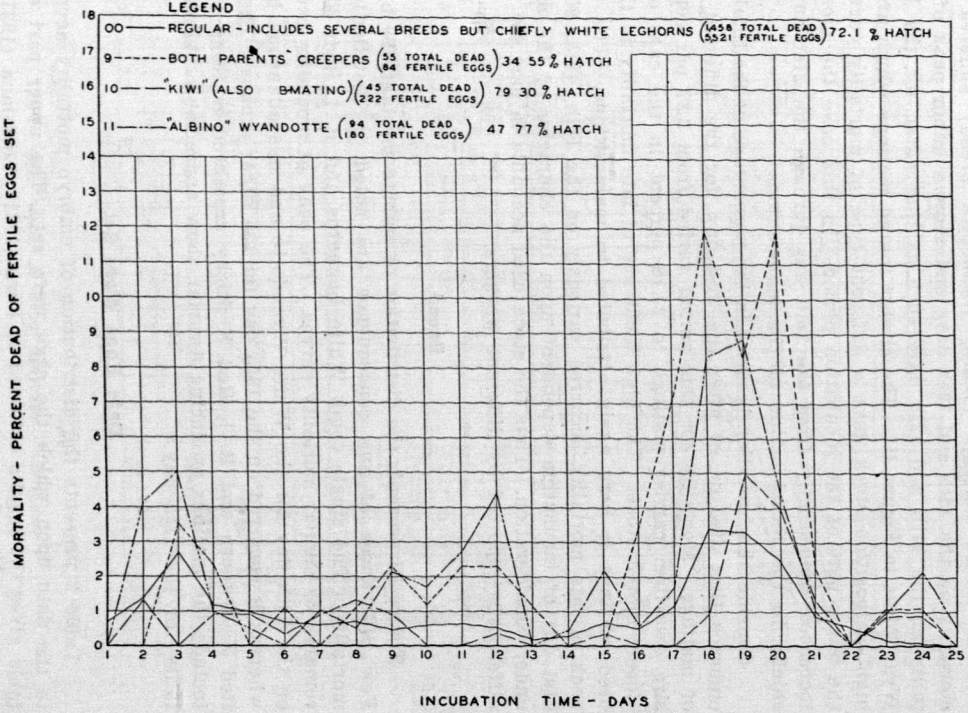


FIGURE II

EMBRYONIC MORTALITY - BY CHARACTER OF MATING (CONTINUED)



that time. The creeper matings showed a somewhat greater mortality for the 8th to 13th days and very high mortality on the 18th, 19th, and 20th days. The peak of mortality at the middle of the incubation period is believed to be due to environmental conditions, while the late peak is due to the death of homozygous creeper embryos.

The albino matings (White Wyandotte breed) suffered high mortality on the 18th and 19th days and show a minor peak of mortality on the 11th and 12th days such as that noted for the White Wyandottes in Figure III. The closely inbred matings which are also high malposition stock gave a different type of mortality curve for the latter part of the incubation period. In this case the mortality increased consistently from the 16th day through the 21st day due chiefly to the lethal action of the malpositions.

Considerable variation is noted in the percentage of the total dead embryos that died on or after the 21st day, for the different types of matings. Mortality for this period varied from 2.27 per cent for sire dwarfism carrier matings to 24.12 percent in the case of the closely inbred stock. The high incidence of late mortality in the inbred matings is here again attributed to malpositions. The major peak of late mortality occurred variously on the 18th, 19th, 20th or 21st day of incubation dependent upon the character of the mating under consideration. Thus the stock used accounts in part for minor differences reported by different workers.

Breed

Figure III presents the distribution of embryo mortality by breeds. Few differences of any consequence are noted in distribution of mortality. The single comb White Leghorns and R. I. Reds yielded remarkably similar mortality curves. The peak of mortality occurred on the 20th day for the White Wyandotte and miscellaneous breeds whereas it occurred on the 18th day for the other groups. The R. I. Red X Leghorn and R. I. Red X Sticky crossbred matings (which include the matings producing dwarfs) show appreciably higher mortality for the 18th day.

Date Eggs Were Set

Table 2 presents the distribution of embryo mortality according to the date upon which the eggs were set. The upper part of the table gives the results obtained at Iowa (1931), Louisiana (1931, 1932 and 1933). The lower part presents data for Iowa (1933). The date the eggs were set was not available for the Iowa (1932) group.

Examination of the data in table 2 reveals that no consistent seasonal difference in distribution of mortality was obtained in either group. This is contrary to the work of Smith (1933) who reported for

FIGURE III

EMBRYONIC MORTALITY - BY BREEDS

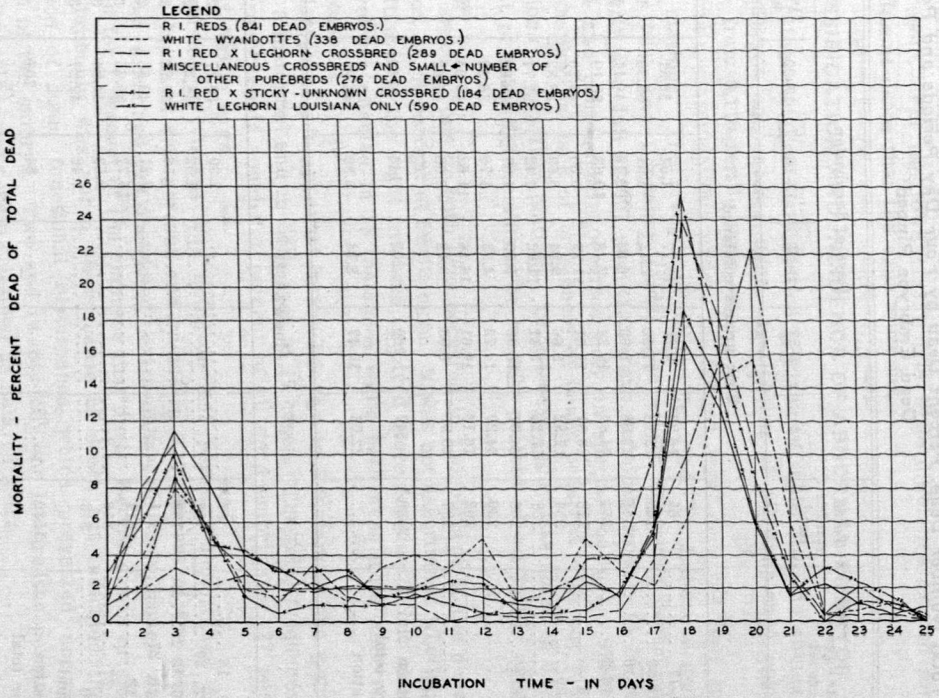


TABLE 2

Total Number Dead, Percent Dead by Four Day Periods and Percent of Dead Embryos Pipped

Date eggs were set	Total number dead	Days of incubation						Percent of total dead pipped
		1-4	5-8	9-12	13-16	17-20	21st and after	
Four groups combined								
January 1-17	65	16.92	9.23	10.77	1.54	52.31	9.23	21.54
January 18-30	165	12.12	11.52	2.42	3.64	59.39	10.91	15.76
February 1-16	347	19.88	8.93	6.63	3.46	46.11	14.99	12.68
February 18-27	254	24.80	10.24	7.48	10.63	38.19	8.66	4.72
March 1-13	278	23.91	10.51	11.23	5.80	44.20	4.35	8.27
March 18-30	254	17.32	8.66	9.84	6.69	48.82	8.66	8.66
April 1-14	228	17.26	8.41	11.06	4.42	51.77	7.08	10.53
April 15-29	268	24.07	13.33	5.19	4.07	48.89	4.44	8.21
May	230	34.20	14.72	4.33	4.76	36.36	5.63	5.65
Sept.-Oct.	202	16.16	10.61	14.14	11.62	43.94	3.54	8.91
Nov.-Dec.	197	29.95	8.63	8.12	5.08	41.62	6.60	8.63
No. for total examination	2488	547	260	202	144	1142	193	235
Percent for total examination	.....	22.03	10.47	8.14	5.80	45.80	7.77	9.45
Iowa—1933								
January 9, 16	87	.....	.....	.....	2.30	59.77	28.73	24.14
January 23, 30	197	.....	.....	.....	1.52	48.22	26.90	25.88
February 6, 13	128	.....	.....	.....	1.56	49.22	21.87	28.12
February 20, 27	111	.....	.....	.....	7.21	49.55	25.22	23.42
March 6, 13	317	.....	.....	.....	15.77	35.65	35.33	22.08
March 20, 27	356	.....	.....	.....	2.53	44.94	32.58	27.81
April 3, 10	343	.....	.....	.....	1.46	49.27	30.61	28.28
No. for total examination	1539	.....	.....	.....	34	707	476	399
Percent for total examination	.....	.....	.....	.....	2.21	45.94	30.93	25.93

Ontario, Canada, a striking difference in seasonal distribution of mortality. This lack of agreement may be due to differences in geographical location and attendant environmental conditions.

The percentage of the total number of dead embryos that pipped for the upper group in table 2 was somewhat higher during January and February than for other months but no consistent seasonal trend was present. For the Iowa (1933) data little difference existed for the different dates that the eggs were set.

CLASSIFICATION AND INCIDENCE OF ABNORMAL EMBRYOS

A rather detailed classification is given in table 3 of the anomalous embryos encountered upon examination of all unhatched eggs (2,488) from 9,110 fertile eggs set.

Certain classifications under "malformed legs" occurred only in a few matings. The creeper character described by Landauer and Dunn (1930) and Landauer (1932), and sticky character described by Byerly and Jull (1930) and (1932) are definitely known to be inherited and consequently occurred only in special matings. Short thick shanks and lack of brittleness of bones occurred with but few exceptions in dwarfism matings. Chondrodystrophy was noted in 6.55 percent of total dead embryos or as 1.79 percent of all fertile eggs set.

Absence of one or both eyes occurred in 2.21 percent of the dead embryos, or .60 percent of fertile eggs. It was not ascertained whether the left eye or the right eye was involved more frequently but only one eye was affected in 35 instances while both eyes were affected in 20 cases.

Microphthalmia appeared in the left eye in 5 instances, the right eye in 3 instances and in both eyes in 17 cases. The total frequency of microphthalmia equaled 1 percent of all dead embryos or .27 percent of fertile eggs.

Eversion of viscera was noted in 2.61 percent, hemorrhage of various kinds in 6.67 percent and edema in 3.17 percent of the dead embryos examined. Constricted extra-embryonic membranes accounted for 1 percent of the embryos that died. Retarded size of embryos probably was not a direct cause of death but indicated the presence of some functional disturbance which prevented normal growth of the embryo. Two and twenty-four one hundredths percent of the unhatched eggs were classified as delayed hatch.

Abnormalities of the head exclusive of eye abnormalities fall chiefly into two categories, abnormal beaks and abnormal skull development or exencephaly. The total so classified is 2.91 percent of the dead embryos of which 1.04 percent is exencephaly, 1.28 percent beak abnormalities, .12 percent head absent and the balance unclassified.

TABLE 3

Classification and Incidence of Abnormal Embryos Encountered

All dead embryos (2,488) examined from 9,110 fertile eggs

Classification of abnormalities	Incidence		
	Number	As percent of total dead embryos	As percent of total fertile eggs set
Chondrodystrophy	163	6.55	1.79
Creepers	33	1.33	.36
Poorly calcified shanks	11	.44	.12
Stickys	17	.68	.19
Short thick shanks	124	4.98	1.36
Legs incompletely formed	6	.24	.07
Unclassified leg abnormalities	8	.32	.09
Total malformed legs	362	14.54	3.98
Absence of one or both eyes	55	2.21	.60
Microphthalmia (one or both eyes)	25	1.00	.27
Unclassified eye abnormalities	4	.16	.04
Total abnormal eyes	84	3.37	.91
Ectopia or eversion of viscera	65	2.61	.71
General internal hemorrhage	67	2.69	.74
General hemorrhage at pipping	19	.76	.21
Hemorrhage at heart	34	1.37	.37
Hemorrhage at liver-kidneys	34	1.37	.37
Hemorrhage at brain	12	.48	.13
Total hemorrhage	166	6.67	1.82
General edema	27	1.08	.30
Enlarged musculus complexus	29	1.17	.32
Edema around head-throat	23	.92	.25
Total edema	79	3.17	.87
Constricted membrane around the body	5	.20	.05
Constricted membrane around head-neck	15	.60	.16
Constricted membrane around legs	5	.20	.05
Total constricted extra-embryonic membranes	25	1.00	.26
Retarded size (for relative development)	45	1.81	.49
Delayed hatch—one day	3	.12	.03
Delayed hatch—two days	19	.76	.21
Delayed hatch—three days	20	.80	.22
Delayed hatch—four days	7	.28	.08
Delayed hatch—number days not specified	7	.28	.08
Total delayed hatch	56	2.24	.62
Color of yolk bright green	21	.84	.23
Ruptured yolk sac	16	.64	.18
Yolk excluded	7	.28	.08
Total yolk abnormalities	44	1.76	.49
Prognathism	10	.40	.11
Upper mandible absent (hyperencephaly)	12	.48	.13
Cross beaks	10	.40	.11
Exencephaly	26	1.04	.29
Head absent (otocephaly)	3	.12	.03
Unclassified head abnormalities	12	.48	.13
Total abnormalities of head[1]	73	2.92	.80

[1] Exclusive of eye abnormalities. See this classification given separately.

TABLE 3 (Continued)

Classification and Incidence of Abnormal Embryos Encountered
Together with a comparison with previous results

Classification of abnormalities	Incidence		
	Number	As percent of total dead embryos	As percent of total fertile eggs set
Twisted spine	2	.08	.02
Incomplete twinning (duplicity)	1	.04	.01
Unclassified Terata	16	.64	.18
Total *miscellaneous*	19	.76	.21
*Embryos too decomposed to permit identification	102	4.10	1.02
Malpositions:			
1. Head between thighs	116	4.66	1.27
2. Head in small end of egg	170	6.83	1.87
3. Head to left	59	2.37	.65
4. Embryo rotated in shell	100	4.02	1.10
5. Head not under wing	93	3.74	1.02
6. Positions 3 and 5 combined	5	.20	.05
7. Positions 4 and 5 combined	15	.60	.16
8. Positions 1 and 2 combined	11	.44	.12
9. Feet over head	2	.08	.02
Total all malpositions	571	22.94	6.26
Total all abnormalities	1589	63.79	17.42

* Not included in total abnormalities.

B.—Comparison of incidence of certain monsters and abnormal conditions with some previous results—As percent of total dead embryos

Classification of abnormalities	Hutt and Greenwood (1929) Edinburgh 11,797 dead	Hutt (1930) Minnesota 5,974 dead	Taylor, Gunns and Moses (1933) 3,448 dead[1]	Present study
Hyperencephaly	1.52	0.13	.....	0.48
Microphthalmia (including eyes absent)	1.06	1.36	1.02	3.21
Exencephaly	1.09	0.25	0.41	1.04
Prognathism	0.07	0.07	0.61[2]	0.40
Ectopia (or eversion of viscera)	0.05	0.10	0.49	2.61
Otocephaly	0.01	0.12	.....	0.12
Legs incomplete	0.02	0.05	.....	0.24
Hemorrhage	.....	.....	1.74	6.67
Edema	.....	.....	1.68	3.17
Duplicity	0.06	0.07	0.06	0.06

[1] These embryos were from chilled eggs but gave quite similar results to 1,308 check lot eggs.

[2] Includes both hyperencephaly and prognathism.

Malpositions constituted 22.94 percent of all dead embryos or 6.26 percent of the fertile eggs set.

The total of 63.79 percent abnormal embryos of all dead or 17.42 percent of all fertile eggs set is slighty higher than actually encountered because a few embryos showed more than one abnormality. This total is greater than obtained by previous investigators. In experiments conducted to ascertain the effects of electric current interruption upon embryo mortality Taylor and coworkers (1933) obtained a total of 33.94 percent anomalous embryos in their control lot.

Hutt and Greenwood (1929) reported that monsters accounted for "at least 3.6 percent of all mortality," Hutt (1930) found 1.07 percent to 4.63 percent terata and he quotes the work of Miss Alsop (1919) in which she identified 6.43 percent abnormally developed embryos, and Byerly (1930) who obtained 8.1 percent terata. The figures are all below the 10.22 percent abnormally formed embryos found in this study. The total (10.22%) includes only embryos with incompetely formed legs, abnormal eyes, eversion of viscera, abnormalities of the head and miscellaneous terata. It does not include chondrodystrophy, malpositions, edema, etc.

In part B of table 3 is given a comparison with previous work, of the incidence of eight types of monsters encountered in this study. Hyperencephaly is the only type of monster that was found less frequently in this study (0.48 percent) than in the previous investigations. Its incidence was greater in Hutt and Greenwood's (1929b) Edinburgh work (1.52 percent), but in the Minnesota work of Hutt this type of monster was much less common (0.13 percent). Microphthalmia and ectopia were much more frequently found in the present study than in the work of Hutt (1930a) or Taylor and coworkers (1933). Hemorrhage and edema were also more common in these data than in Taylor's.

Eye and brain defects constituted only 47.85 percent of total monsters in this work but comprised 93 percent of all terata in Hutt and Greenwood's data and 74 percent in Byerly's (1930) data.

One hundred and two embryos, or 4.10 percent of the total number examined, were in such a state of decomposition that it was not possible to determine whether or not any abnormality existed.

Incidence of Certain Abnormalities—Character of Mating

The incidence of the commonly encountered abnormalities, was determined for matings of different character. These data are presented in table 4.

Chondrodystrophy occurred much more frequently in the albino Wyandotte matings (23.40 percent of all dead embryos) than in any other type of mating. Kiwi matings also show high incidence of this condition. The incidence of chondrodystrophy obviously varied widely for the different types of matings.

TABLE 4

Incidence of Certain Abnormalities—By Character of Mating

Percent of Total Dead Embryos

Character of mating	Total No. dead	No. fertile eggs set	Chondrodystrophy	Absence of, and micro-eyes	Eversion of viscera	Hemorrhage	Edema	Abnormal membranes	Retarded size	Delayed hatch	Yolk abnormalities	Beak abnormalities	Exencephaly	Total abnormalities including malpositions (See table 9)
0 Regular	1458	5521	4.87	3.77	3.22	7.41	4.05	1.10	2.33	.69	1.78	1.85	1.85	53.29
1 Mildly Inbred	211	983	9.48	3.32	1.90	4.74	2.37	1.52	1.42	8.53	1.42	.95	.....	72.62
3 Sire Dwarf Carrier..	132	481	9.09	5.30	1.52	5.30	1.52	1.73	.....	2.27	2.27	.76	1.52	57.81
4 Both Dwarf Carriers.	289	794	4.15	3.81	1.73	3.81	1.38	.....	1.73	1.04	1.73	.....	1.04	50.17
9 Both Creeper	55	84	5.45	3.64	1.82	5.45	.....	.....	.....	3.64	1.82	3.64	.....	47.27
10 "Kiwi" Mating	45	222	15.56	.....	6.67	4.44	2.22	.....	.....	13.33	.....	.....	.....	77.77
11 Albino Wyandotte ...	94	180	23.40	1.06	.....	14.89	3.19	2.13	.....	14.89	1.06	.....	.....	85.09
15 Sire Sticky—Both Dwarf Carriers	115	225	6.07	.87	3.48	4.35	3.48	.....	.87	.87	2.61	.....	.87	54.78
Miscellaneous Matings ...	89	620	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
Average All Matings.....	2488	9110	.....	3.33	2.61	6.67	3.17	1.00	1.81	2.24	1.76	1.28	1.04	.....

Eye abnormalities were somewhat more frequent in the sire dwarf carrier matings but occurred rather uniformly in all types of matings. Hemorrhage occurred most frequently in the Albino Wyandotte matings (14.98 percent of all dead embryos) but was rather evenly distributed in the other types of matings. Delayed hatch was confined chiefly to three types of matings. It accounted for 14.89 percent of Abino Wyandotte dead, 13.33 percent of kiwi dead and 8.53 percent of the mildly inbred; yet occurred as only 2.42 percent of all dead embryos. The other abnormalities given in table 4 do not appear to be associated with any certain type of mating. A total of 72.62 percent of the mildly inbred, 77.77 percent of the kiwi, and 85.09 percent of the Albino Wyandotte matings were classified as having some abnormality. For the five other types of matings only about one-half of all dead embryos were classified as abnormal.

Incidence of Abnormalities—Breed

Distribution of the various abnormal embryos encountered is given in table 5 by breeds.

Excluding the miscellaneous group, chondrodystrophy was much more common in the White Wyandottes used than in any other breed. Malpositions accounted for 46.19 percent of all dead embryos in the highly inbred single comb White Leghorns, 35.87 percent of the miscellaneous group, and approximately 25 percent for White Wyandottes, Red X Leghorn crossbreds and Red X sticky crossbreds. The regular single comb White Leghorn group had 21.76 percent malpositions and the R. I. Reds only 15.11 percent so classified. The more rarely occurring abnormalities were practically all found to some extent in the different breeds although varying considerably in frequency of occurrence. Hemorrhage and edema were more common in the White Wyandotte embryos and exencephaly and retarded size were more common in the Rhode Island Reds.

Some types of abnormalities, notably chondrodystrophy, hemorrhage, malpositions and delayed hatch varied widely in occurrence for matings of different types and for different breeds. Certain malpositions varied from year to year and for the two institutions as did the frequency of total malpositions. It was believed and later analysis shows that these differences in incidence of abnormalities were not accidental.

Incidence of Abnormalities—Date Eggs Were Set

The data in table 6 are arranged to reveal the incidence of various abnormalities resulting from eggs set at different dates. The total number of dead embryos is given for each date upon which eggs were set. The upper part of the table gives the data for four groups combined, namely: Iowa (1931), Louisiana (1931), Louisiana (1932), and Louisiana (1933). The occurrence of chondrodystrophy in the Iowa (1933) group is given in the lower part of the table.

TABLE 5

Incidence of Certain Abnormalities—By Breeds

Percent of Total Dead Embryos

Breeds	Total No. of dead embryos	Chondrodystrophy	Malpositions							
			1	2	3	4	5	9	Total No.	Total %*
Miscellaneous Breeds and Crossbreds.	276	11.96	9.78	9.42	7.25	3.62	6.16	.36	99	35.87
White Leghorn	593	5.73	4.22	8.10	.34	6.74	4.72	0.00	129	21.76
R. I. Red	841	3.21	2.74	5.11	2.14	2.74	2.62	.12	127	15.11
White Wyandotte	338	12.72	8.88	6.22	3.55	6.51	2.97	0.00	89	26.34
Crossbred Red and Leghorn	289	4.15	4.50	7.96	1.73	4.50	10.04	0.00	81	28.03
Crossbred Red and Sticky	184	7.07	4.34	10.87	3.80	3.80	3.25	0.00	45	24.44
Highly Inbred Leghorns	2429	3.80	3.29	6.42	9.02	2.88	22.19	2.39	1122	46.19

TABLE 5 (Continued)

Incidence of Certain Abnormalities—By Breeds

Percent of Total Dead Embryos

Breeds	Total No. of dead embryos	Abnormal eyes	Eversion of viscera	Hemorrhage	Edema	Abnormal membranes	Retarded size	Delayed hatch	Yolk abnormalities	Beak abnormalities	Exencephaly	Embryo too decomposed	Pipped
Miscellaneous Breeds and Crossbreeds	276	3.26	1.81	5.80	2.17	1.45	1.81	2.54	2.17	1.45	1.09	4.35	11.96
White Leghorn	593	3.54	2.70	6.07	2.19	.17	1.01	2.36	1.69	1.69	.34	2.53	8.69
R. I. Red	841	3.92	3.92	4.16	3.21	.59	3.09	1.66	1.78	1.55	2.26	7.73	9.27
White Wyandotte	338	1.80	.89	13.02	7.40	2.37	.30	2.07	1.18	1.78	.30	2.66	3.55
Crossbred Red and Leghorn	289	3.46	1.38	3.46	.69	1.73	1.38	1.69	1.73	.69	.69	.69	14.53
Crossbred Red and Sticky	184	.54	2.17	3.80	3.26	0.00	1.09	1.19	2.17	.54	.54	1.63	10.33
Highly Inbred Leghorns	2429	....	....	.....	....	....	....	....	....	....	....	....	.....

* Total percents do not correspond to individual percents added since some malpositions are counted twice because two positions were shown by the same embryo.

TABLE 6

Incidence of Various Abnormalities—By Date Eggs Were Set

Percent of Total Dead Embryos

Date eggs were set	Chondrodystrophy	Absent and microphthalmia eyes	Eversion of viscera	Hemorrhage	Edema	Abnormal membranes	Retarded size	Delayed hatch	Yolk abnormalities	Beak abnormalities	Exencephaly	Total number dead	Total abnormalities
Four groups combined[1]													
January 1-17	9.23	0.00	1.54	14.04	1.54	0.00	0.00	0.00	0.00	3.08	0.00	65	64.83
January 18-30	7.88	.61	6.67	6.45	3.03	0.00	.61	3.64	2.42	1.21	1.21	165	54.94
February 1-16	5.19	1.15	2.88	5.79	2.88	.29	3.75	7.20	1.15	.86	2.02	347	54.19
February 18-27	4.33	1.97	1.18	10.92	7.48	1.97	2.36	1.18	.39	.79	.79	254	53.05
March 1-13	6.83	2.52	2.52	9.02	2.88	4.68	1.44	1.44	2.52	.72	.72	278	61.19
March 18-30	11.81	3.54	1.18	4.96	3.15	1.97	.79	2.76	4.33	.39	0.00	254	64.79
April 1-14	7.46	3.51	2.19	8.06	3.07	1.75	1.32	.44	0.00	1.32	0.00	228	66.40
April 15-29	5.60	4.48	1.87	7.20	1.12	.75	1.49	1.49	1.49	1.49	1.12	268	54.22
May (Including one June hatch)	8.26	7.39	2.68	2.68	.87	.87	2.17	.87	1.30	.87	.87	230	46.64
Sept.-Oct.	2.48	5.45	5.94	4.12	3.47	.50	1.98	0.00	1.98	4.46	3.47	202	49.21
Nov.-Dec.	8.12	5.08	1.03	4.23	5.58	.51	3.05	1.02	3.05	1.52	.51	197	54.51
Total number examined..	169	84	65	154	79	34	48	55	44	33	26	2488	
Iowa 1933													
January 9, 16	3.60	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	87	.....
January 23, 30	0.00	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	197	.....
February 6, 13	0.00	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	128	.....
February 20, 27	6.31	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	111	.....
March 6, 13	2.84	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	317	.....
March 20, 27	4.21	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	356	.....
April 3, 10	2.62	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	343	.....
Number for total examination	44	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	1539	.....
Percent for total examination	2.86	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....

[1] Iowa 1931, Louisiana 1931, 1932 and 1933.

Chondrodystrophy has been previously reported to vary according to the time of the year that the eggs are incubated. This tendency is not present in the data of this study. Using the method of Snedecor and Irwin (1933), a test for homogeneity was made of the occurrence of chondrodystrophy for the four combined groups. This test gave a chi-square of 22.909 or P of between 0.02 and 0.01, indicating that the sub-samples are probably not uniform in regard to the occurrence of this condition. Nevertheless no sensible seasonal trend is present. The same holds true of the Iowa (1933) data.

Hutt and Greenwood (1929) and Munro (1932) report that the incidence of chondrodystrophy decreased as the season advanced but Byerly, Titus and Ellis (1933a) hold that seasonal distribution "might be accounted for in other ways." The latter workers concluded that some nutritional deficiency other than vitamin D is the underlying cause of this condition. Their hypothesis is based upon the increase in incidence of chondrodystrophy for birds on diets containing proteins from vegetable sources. However these same workers have shown that only certain hens were affected and that distribution of chondrodystrophy cannot be attributed to chance.

Abnormalities of the eyes, on the other hand, revealed a marked seasonal trend, becoming more frequent as the season advanced (Figure IV). Correlation of the date eggs were set and incidence of eye abnormalities gave a coefficient of $r = + .927$; with .708 required for a high degree of significance. This is especially true for the settings from January through May. Why this type of terata showed such a seasonal difference when other classes of anomalies did not vary with the season, is not known. Further data are needed to verify this tendency. The incidence of hemorrhage decreased as the year advanced but the trend is not so pronounced. A coefficient of $r = — .690$ was found when date eggs were set and incidence of hemorrhage were correlated. A coefficient of .708 is required for a high degree of significance although $r = .576$ or larger gives reasonable assurance of association in this case. It may be stated then that hemorrhage occurred less often as the spring season advanced but the association was not close and is of questionable significance.

The percentage of abnormal membranes increased during January and February, was the highest in March 1 to 13 settings then decreased consistently toward the end of the year. However, the number of this class of abnormalities was not great at any time. Delayed hatch embryos were encountered much more frequently in eggs set during the latter part of January and the first of February, while excencephaly was most common in the September-October settings. None of these abnormal types showed any consistent seasonal trend except the two instances mentioned.

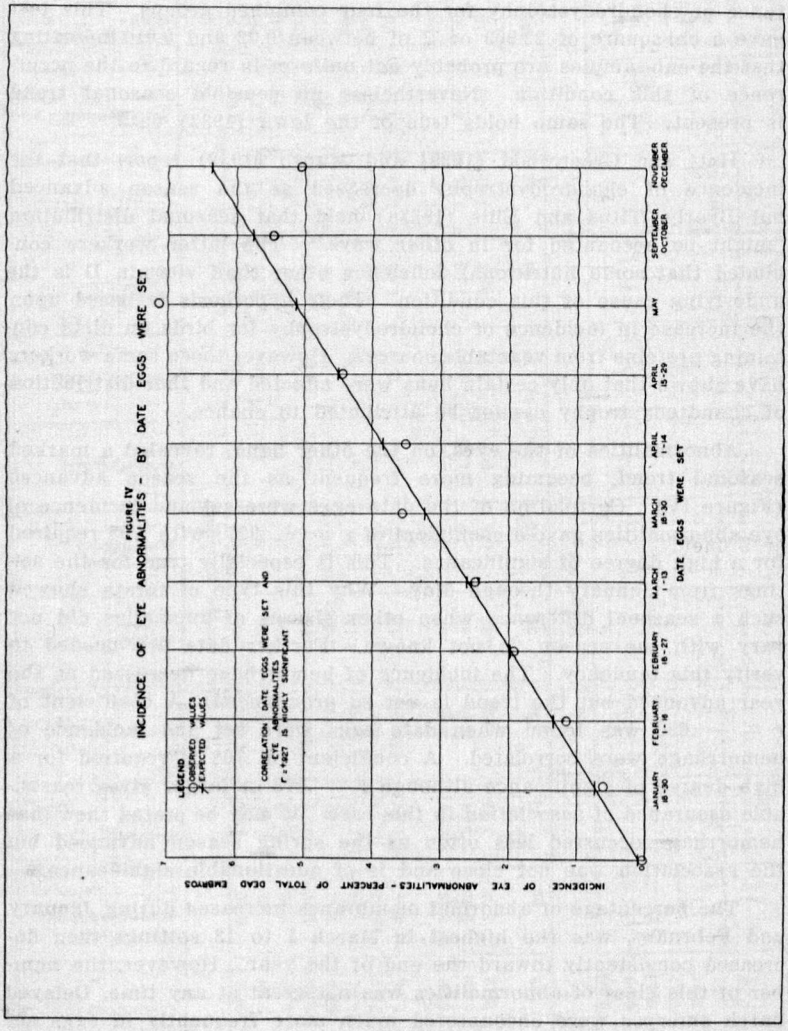


FIGURE IV
INCIDENCE OF EYE ABNORMALITIES - BY DATE EGGS WERE SET
LEGEND
OBSERVED VALUES
EXPECTED VALUES
CORRELATION, DATE EGGS WERE SET AND EYE ABNORMALITIES r = .9827 IS HIGHLY SIGNIFICANT
INCIDENCE OF EYE ABNORMALITIES - PERCENT OF TOTAL DEAD EMBRYOS
7
6
5
4
3
2
1
0
JANUARY 16-30
FEBRUARY 1-15
FEBRUARY 16-27
MARCH 1-13
MARCH 16-30
APRIL 1-14
APRIL 15-29
MAY
SEPTEMBER OCTOBER
NOVEMBER DECEMBER
DATE EGGS WERE SET

POSSIBLE INCIDENCE OF CERTAIN ABNORMALITIES

The incidence of abnormalities encountered for the groups in which all dead embryos were examined was determined by sires. Upon examination of the data some striking differences were noted for the different sires. This suggested that some of the conditions observed might be inherited.

Periodic Mortality, and Certain of the More Commonly *Noted Abnormalities*

The first condition to be considered (table 7) is the distribution of mortality at certain periods for the different sires. The particular periods considered are mortality on first to fifth days of incubation, on 17th to 21st days of incubation and mortality on 22nd day and thereafter. It may be noted that this last classification corresponds to delayed hatch mentioned in earlier sections. The percentages of fertile eggs set that were classified as abnormal eyes, eversion of viscera, edema, hemorrhage and dead embryos pipped, are also given in table 7. The incidence of hemorrhage and percent of dead embryos pipped is given by sires in table 8 for the males used in the Iowa (1932) and Iowa (1933) matings.

Eight sires were used for two seasons, making it possible to test the influence of these males for different years. These sires were not mated to the same hens for the two years in many cases but important influences of the sire upon embryo mortality might be in evidence. In five of the eight cases the mortality of embryos of the same sire during the first five days of incubation was in close agreement for the two years. However, in three instances, this early embryonic mortality increased noticeably the second year. In all cases except two (sire 1727 and sire 2125) the 17-21 day mortality was greater for the second year mated. Little consistency is noted in the mortality for the 22nd day and thereafter for the same sire in different years. Sire 2770 produced the largest percent of dead embryos classified as delayed hatch (3.23 percent). The two sires with greatest mortality for the 17-21 day period are 4016 with 34.01 percent dead and 3719 with 27.61 percent dead. Sire 4016 was a carrier of the lethal gene, stickiness, and 3719 was a creeper male. Four of the seven other males with 19.23 percent or more dead embryos in the 17-21 day period, sired dwarfism matings, two sired inbred matings and one sired the Albino Wyandotte mating. More specific analysis is made of the mortality in these matings in a later section.

The incidence of abnormal eyes was greatest in the progenies of sire 99 in 1932, and sire 8118 in 1932, however it was not consistent for the same sire in different years. While the distribution did not occur at random no simple relations were found. The same is true of

TABLE 7

Periodic Mortality and Certain of the More Commonly Noted Abnormalities
By Sires

Year and institution used	Band numbers of sires	Number fertile eggs set	Percent of fertile eggs set								
			Days of incubation			Abnormal eyes	Eversion of viscera	Edema	Hemorrhage* percent of fertile eggs set	Pipped	
			1-5 days	17-21 days	22 and[1] after					Percent of total dead embryos	Percent of fertile eggs set
Iowa 1931	1727	305	5.25	16.39	.66	.98	.33	3.93	7.21	0.00	0.00
Louisiana 1932	1727	110	13.64	15.45	0.00	0.00	.91	0.00	0.00	0.00	0.00
Louisiana 1933	1727	21	.....	.....	.....	.....	.....	.....	.....	.....	.....
Iowa 1931	2505	117	11.11	14.53	0.00	.85	.85	2.56	5.98	0.00	0.00
Iowa 1931	5124	156	4.49	11.54	.64	1.92	0.00	0.00	6.41	2.22	.64
Louisiana 1931	99	121	10.74	6.61	0.00	4.13	.83	2.48	0.00	2.78	.83
Louisiana 1932	99	220	14.09	10.45	2.27	.91	1.36	0.00	.91	13.00	4.09
Louisiana 1931	100	164	8.54	11.59	.61	2.44	3.05	1.22	.61	6.12	1.83
Louisiana 1932	100	320	7.81	19.67	2.19	.94	1.25	1.25	1.25	14.15	4.69
Louisiana 1932	6	160	12.50	10.00	.63	.62	.63	0.00	.63	12.50	3.75
Louisiana 1932	456	277	6.86	5.78	1.08	.36	0.00	.36	.36	2.38	.36
Louisiana 1933	456	182	13.19	19.23	1.10	0.00	.55	.55	1.10	17.65	6.59
Louisiana 1932	2125	360	6.67	8.06	1.67	.28	.56	.28	1.11	10.29	1.94
Louisiana 1933	2125	102	13.73	.98	0.00	0.00	0.00	0.00	1.96	0.00	0.00
Louisiana 1932	2770	270	5.93	4.81	.74	1.11	.37	.37	.37	5.26	.74
Louisiana 1933	2770	186	2.69	12.90	3.23	0.00	0.00	0.00	.54	14.29	2.69
Louisiana 1933	97	155	9.03	23.23	.65	1.29	.65	1.29	1.94	7.25	3.23
Louisiana 1933	155	186	8.06	23.66	3.76	.54	0.00	1.61	7.53	11.70	5.91
Louisiana 1933	3261	586	8.87	22.87	.51	2.22	.34	.17	2.22	12.67	4.78
Louisiana 1933	3719	134	4.48	27.61	1.49	1.49	.75	0.00	2.24	18.97	8.21
Louisiana 1933	3930	270	4.81	7.41	2.96	.37	.74	.74	.74	16.33	2.96
Louisiana 1933	4016	344	7.85	34.01	1.16	.29	1.16	1.74	2.03	11.30	5.81
Louisiana 1933	4144	113	7.96	7.08	0.00	0.00	0.00	0.00	.88	15.79	2.65
Louisiana 1933	4597	241	0.00	14.94	.41	0.00	.41	.41	.41	11.90	2.07
Louisiana 1933	4705	144	2.78	9.72	0.00	0.00	.69	.69	.69	10.53	1.39
Louisiana 1933	4740	150	2.00	21.33	0.00	.67	0.00	0.00	3.33	9.76	2.67
Louisiana 1933	4790	184	2.72	13.59	1.63	0.00	1.63	.54	1.09	13.95	3.26
Louisiana 1932	4974	76	9.21	1.32	1.32	1.32	1.32	0.00	1.32	0.00	0.00
Louisiana 1933	4974	118	7.63	3.39	0.00	3.39	0.00	0.00	.85	0.00	0.00
Louisiana 1932	8118	64	10.94	7.81	0.00	6.25	1.56	0.00	1.56	7.14	1.56
Louisiana 1933	8118	218	13.76	23.39	1.38	.46	1.38	.92	1.83	10.00	4.59
Test of Homogeneity	X^2				49.16	71.35	33.22	72.59			
	P				Between .02-.01	Less than .01	Between .30 & .20	Less than .01			

[1] This period corresponds to "Delayed Hatch."

* Homogeneity test yields a chi-square of 128.33 and P less than .01.

TABLE 8

Incidence of Hemorrhage and Percent of Dead Embryos Pipped

By Sires

(Of Iowa 1932 and Iowa 1933 Groups)

Institution and year used	Band numbers of sires	Number fertile eggs set	Total number dead embryos	Breed[1]	Hemorrhage[2] percent of fertile eggs set	Pipped	
						Percent total dead embryos	Percent fertile eggs set
Iowa 1932	438	274	26	I. Wh. L.	0.73	0.00	0.00
Iowa 1932	1467	315	143	"	1.59	6.99	3.17
Iowa 1932	1518	293	70	"	4.44	4.29	1.02
Iowa 1932	2533	435	225	"	9.89	4.89	2.53
Iowa 1932	3119	379	68	"	2.37	4.41	0.79
Iowa 1932	3191	459	80	"	2.61	3.75	0.65
Iowa 1932	3214	235	93	"	1.70	3.23	1.28
Iowa 1932	4962	343	127	"	4.08	3.94	1.46
Iowa 1933	105	536	154	"	.....	20.78	5.97
Iowa 1933	111	255	60	?	.....	21.67	5.10
Iowa 1933	112	451	170	I. Wh. L.	.....	21.76	8.20
Iowa 1933	126	400	220	"	.....	16.81	9.25
Iowa 1933	135	440	188	"	.....	14.36	6.14
Iowa 1933	170	740	134	"	.....	33.58	6.08
Iowa 1933	204	532	178	"	.....	29.78	9.96
Iowa 1933	202	548	253	"	.....	42.67	19.71
Iowa 1933	488	.....	.....	?	.....	.....	.....
Iowa 1933	3172	539	178	I. Wh. L.	.....	21.91	7.24
Mean	.....	.....	.....	.....	.....	.....	5.209
Standard deviation	.....	.....	.....	.....	.....	.....	1.824

[1] I. Wh. L., Inbred single comb White Leghorn.
? Unknown breeding.
[2] Homogeneity test yields a chi-square of 63.55 and P. less than .01.

TABLE 9

Incidence of Chondrodystrophy, Malpositions, and Sex

By Sires (of Iowa 1931, Louisiana 1931, Louisiana 1932 and Louisiana 1933 groups)

Institution and year used	Band numbers of sires	Number fertile eggs set	Total number dead embryos	Breed[1]	Chondrodystrophy (Percent of fertile eggs set)	Malpositions							Sex			Sex ratio with significance of Chi-square[2]
						1	2	3	4	5	Total number	Percent of 18th day living embryos	Number unknown	Number males	Number females	
Iowa 1931	1727	305	102	Wh. W.	3.61	11	7	3	11	2	34	13.33	26	31	45	40.79
Louisiana 1932 ...	1727	110	45	Wh. W.	4.55	1	0	0	4	1	6	7.50	27	7	11	38.89
Louisiana 1933 ...	1727	21	10	Wh. W.	.....	0	2	0	0	2	4	21.05	0	3	7	30.00
Iowa 1931	2505	117	40	Wh. W.	3.42	4	2	2	1	1	10	10.75	20	6	14	30.00*
Iowa 1931	5124	156	45	Wh. W.	0.00	8	3	4	2	1	18	13.95	13	19	13	58.06
Louisiana 1931 ...	99	121†	36	R. I. R.	1.65	0	1	1	0	0	2	2.22	21	4	11	26.67
Louisiana 1932 ...	99	220†	69	R. I. R.	0.00	0	1	3	2	0	6	3.39	44	13	12	52.00
Louisiana 1931 ...	100	164†	49	R. I. R.	.61	2	3	1	1	1	8	6.11	22	9	18	33.33*
Louisiana 1932 ...	100	320†	106	R. I. R.	.31	0	6	5	7	1	19	6.99	39	38	29	56.72
Louisiana 1932 ...	6	160†	48	R. I. R.	.63	2	3	0	0	0	5	3.91	30	9	9	50.00
Louisiana 1932 ...	456	277	42	R. I. R.	0.00	1	3	0	1	1	6	2.42	24	8	10	44.44
Louisiana 1933 ...	456	182	68	R. I. R.	2.20	1	6	0	4	5	16	10.74	31	17	20	45.95
Louisiana 1932 ...	2125	360	68	Wh. L.	.83	2	7	0	2	0	11	3.43	32	18	18	50.00
Louisiana 1933 ...	2125	102	15	Wh. L.	0.00	0	0	0	0	1	1	1.14	14	0	1	.....
Louisiana 1932 ...	2770	270	38	Wh. L.	0.00	0	3	1	1	1	6	2.43	24	8	6	57.14
Louisiana 1933 ...	2770	186	35	Wh. L.	.54	2	4	1	2	3	12	6.67	5	10	20	33.33*
Louisiana 1933 ...	97	155	69	I. Wh. L.	4.52	3	4	1	6	9	23	18.85	28	23	18	56.10
Louisiana 1933 ...	155	186	94	A. Wh. W.	11.83	5	8	4	1	5	23	16.43	27	38	29	56.72
Louisiana 1933 ...	3261	586	221	X. B.	2.39	10	20	5	7	29	71	14.37	84	66	71	48.18
Louisiana 1933 ...	3719	134	58	Cr.	2.24	1	4	2	3	3	13	11.93	15	20	23	46.51
Louisiana 1933	3930	270	49	R. I. R.	1.11	2	3	0	6	1	12	4.90	18	19	12	61.29

Louisiana 1933 ...	4016	344	177	St. X. B.	3.78	8	20	7	7	6	48	17.71	48	64	65	49.61
Louisiana 1933 ...	4144	113	19	Wh. L.	.88	1	1	1	1	2	6	5.83	10	4	5	44.44
Louisiana 1933 ...	4597	241	42	Wh. L.	0.00	0	7	3	11	11	32	13.62	1	15	26	36.59*
Louisiana 1933 ...	4705	144	19	Wh. L.	.69	1	4	1	1	1	8	5.88	5	7	7	50.00
Louisiana 1933 ...	4740	150	41	Wh. L.	1.33	4	6	1	2	5	18	13.04	6	24	11	68.57*
Louisiana 1933 ...	4790	184	43	K. X. B.	3.80	5	8	0	1	2	16	9.47	16	13	14	48.15
Louisiana 1932 ...	4974	76	12	I. Wh. L.	0.00	0	0	0	0	0	0	0.00	9	1	2	.....
Louisiana 1933 ...	4974	118	13	I. Wh. L.	0.00	0	1	0	1	1	3	2.75	9	2	2	.....
Louisiana 1932 ...	8118	64	14	R. I. R.	0.00	0	2	0	1	0	3	5.56	7	5	2	.....
Louisiana 1933 ...	8118	218	100	R. I. R.	4.13	8	2	4	4	13	31	18.67	41	27	32	45.76
Means	.....	.....	.....	.....	1.835	....	....	....	....	....	.....	8.873	.....	.....	.....	46.64
Standard Deviation	.....	.....	.....	.....	0.456	....	....	....	....	....	.....	0.997	.....	.....	.....	6.45

[1] Breeds: Wh. W., White Wyandotte; R. I. R., Rhode Island Red; Wh. L., Single comb White Leghorn; I. Wh. L., Inbred single comb White Leghorn; A. Wh. W., Albino White Wyandotte; X. B., Crossbred; Cr., Creeper; St. X. B., Sticky carrier crossbred; K. X. B., Kiwi crossbred.

[2] *—P of between .05 and .01.

† Approximate figures only.

TABLE 10

Incidence of Chondrodystrophy, Malpositions, and Sex

By Sires (of Iowa 1932 and Iowa 1933 groups)

Institution and year used	Band number of sires	Number fertile eggs set	Total number dead embryos	Breed[1]	Chondrodystrophy (Percent of fertile eggs set)	Malpositions								Sex			Sex ratio with significance of Chi-square[2]
						1	2	3	4	5	9	Total number	Percent of 18th day living embryos	Number unknown sex	Number males	Number females	
Iowa 1932..	438	274	26	I. Wh. L.	0.00	1	3	3	1	1	0	9	3.52	15	7	4	63.64
Iowa 1932..	1467	315	143	I. Wh. L.	.32	4	7	1	1	21	0	34	14.66	73	31	39	44.29
Iowa 1932..	1518	293	70	I. Wh. L.	.34	5	5	10	2	10	0	32	12.36	31	14	25	35.90*
Iowa 1932..	2533	435	225	I. Wh. L.	3.68	5	6	24	10	39	0	84	23.86	87	54	84	39.13*
Iowa 1932..	3119	379	68	I. Wh. L.	0.00	6	9	1	9	15	0	40	11.36	24	14	30	31.82*
Iowa 1932..	3191	459	80	I. Wh. L.	.65	4	1	4	2	7	0	18	4.35	39	13	28	31.71*
Iowa 1932..	3214	235	93	I. Wh. L.	5.96	4	5	10	3	5	0	27	15.52	54	16	23	41.03
Iowa 1932..	4962	343	127	I. Wh. L.	3.21	3	4	16	4	16	0	43	16.29	71	18	38	32.14†
Iowa 1933..	105	536	154	I. Wh. L.	.19	10	16	12	3	44	0	85	17.75	53	42	59	41.58
Iowa 1933..	111	255	60	?	0.00	1	4	5	3	12	0	25	10.46	26	16	18	47.06
Iowa 1933..	112	451	170	I. Wh. L.	1.55	3	10	13	3	36	10	75	19.53	65	50	55	47.62
Iowa 1933..	126	400	220	I. Wh. L.	1.00	7	18	19	2	62	17	125	39.06	79	48	93	34.04†
Iowa 1933..	135	440	188	I. Wh. L.	2.27	13	13	14	9	50	5	104	26.60	59	47	82	36.43†
Iowa 1933..	170	740	134	I. Wh. L.	.14	1	16	12	3	50	0	82	11.48	33	39	62	38.61*
Iowa 1933..	204	532	178	I. Wh. L.	.94	7	16	25	4	37	2	91	19.32	60	47	71	39.83*
Iowa 1933..	202	548	253	I. Wh. L.	.36	3	10	18	4	77	22	134	27.40	70	63	120	34.43†
Iowa 1933..	488	.....	.....	?	.....	2	5	10	0	10	0	27	.....	1	12	29	.....
Iowa 1933..	3172	539	178	I. Wh. L.	.37	1	8	22	7	47	2	87	17.54	57	42	79	34.71†
Means	.....	.....	.....		1.234	....	....	....	....	....	....	.....	17.121	.....	.....	.....	39.65
Standard deviations	.....	.....	.....		.414	....	....	....	....	....	....	.....	4.412	.....	.....	.....	1.984

[1] I. Wh. L. Inbred single comb White Leghorn.
? Unknown breed.

[2] Significance of Chi-square:
* P of between .05 and .01.
† Highly significant difference, i. e., P less than .01.

the occurrence of eversion of viscera and edema. If these anomalies are inherited at all the present data are too meager to permit determination of the mode of inheritance.

The average incidence of hemorrhage for the sires considered in table 7 is 1.903 percent of fertile eggs set with a standard deviation of 0.389. The incidence of this class of abnormal embryos (Tables 7 and 8) varied too greatly to be attributed to chance alone (P less than 0.01) but for the sires with the greatest percentage present no simple relations were evident for the various dams to which they were mated.

The percentage of fertile eggs set that was classified as pipped but failed to hatch, varied considerably. An average of 2.703 percent was obtained for the sires considered in tables 7 and 5.209 percent was the average for the highly inbred sires (table 8). A positive correlation existed between percent total malpositions and percent pipped for the sires cited in table 7. The association was not intimate however as indicated by a coefficient of $r = + .491$. This relationship did not exist for the inbred sires of table 8.

Chondrodystrophy

Chondrodystrophy is very unevenly distributed among the various sires of both tables 9 and 10. This led to tests for homogeneity of this characteristic.

A test of the homogeneity of the incidence of chondrodystrophy for all sires that fertilized one hundred or more eggs is given by groups in table 11. All results for a sire used in two or more years are included in the group in which he was originally used.

In four of the six groups (table 11) incidence of chondrodystrophy is definitely different for the various sires. The two remaining groups include two repeat sires in one case and three repeat sires in the other. This fact, together with the low incidence of chondrodystrophy in these groups as a whole, may account for the homogeneous distribution in these cases. When all groups are combined the results indicate that the incidence of chondrodystrophy varied more from sire to sire than can be accounted for by random sampling. If this character were inherited and the several sires differed genetically in regard to it, such results would be expected.

Table 12 presents homogeneity tests for the distribution of chondrodystrophy according to dams mated to certain sires. Only sires producing a higher incidence of chondrodystrophy are included in this table. Part A of table 12 includes Louisiana matings and part B gives Iowa matings.

TABLE 11

Homogeneity of Incidence of Chondrodystrophy According to Sires

By Group

Group	Number of sires	Degrees of freedom	Sires repeated[1]	Number fertile eggs set	Incidence of chondrodystrophy		Chi-square and significance[2]
					Number	Percent	
Iowa 1931.....	5	4	1-3	709	24	3.39	21.67†
Louisiana 1931...	4	3	2-2	825	4	.485	.472
Louisiana 1932...	7	6	3-2	1537	9	.586	9.49
Iowa 1932.....	8	7	0	2733	46	1.68	61.54†
Iowa 1933.....	9	8	0	4441	32	.721	38.49†
Louisiana 1933...	15	14	0	2983	82	2.75	84.90†
Totals	48	42	.....	13,228	197	.....	216.562†

[1] Sires repeated. Those sires used in the later years are included (for all years) in the group in which they originally appear. Example, for the Iowa 1931 group 1-3 means that one sire was used for three years.

[2] † Highly significant differences, i. e., P = less than .01.

TABLE 12

Homogeneity of Incidence of Chondrodystrophy According to Dams

For Certain Sires

Part A (Louisiana Matings)

Band numbers of sires	Number of dams	Degrees of freedom	Number of embryos living at five days	Incidence of chondrodystrophy		Chi-square and significance[1]
				Number	Percent	
97	6	5	168	10	5.95	14.54*
155	6	5	177	23	12.99	11.49*
456	4	3	159	3	1.89	.750
3261	14	13	538	13	2.42	19.42
3719	4	3	119	3	2.52	6.82
4016	6	5	314	13	4.14	6.84
4790	4	3	185	7	3.78	6.30
8118	8	7	188	9	4.79	23.72†
Totals	52	44	1,848	81	.....	89.88†

Part B (Iowa Matings)

Band numbers of sires	Number of dams	Degrees of freedom	Number of fertile eggs set	Incidence of chondrodystrophy		Chi-square and significance[1]
				Number	Percent	
2533	13	12	435	16	3.68	153.99†
3214	11	10	235	14	5.96	46.23†
4962	15	14	343	11	3.21	29.97†
135	15	14	440	10	2.27	20.90
112	13	12	451	6	1.33	20.46
126	16	15	400	4	1.00	29.62*
204	15	14	532	5	.94	34.09†
Totals	98	91	2,836	66	.....	335.26†

[1] According to method of Snedecor and Irwin (1933).

* — P of between .05 and .01.

† — P of less than .01, therefore highly significant differences.

For five of the eight Louisiana sires the distribution of chondrodystrophy according to dams was homogeneous but in all cases the probability of such distribution was rather remote. In the three other cases, which showed a higher incidence of chondrodystrophy, real differences in dams appeared to exist. When data of all Louisiana sires are combined the Chi-square obtained is of such magnitude as to signify that the distribution of chondrodystrophy among all dams was not due to chance variation alone. The same holds true for the combined Iowa matings.

For five of the seven groups of the Iowa matings chondrodystrophy was not normally distributed and for the other two groups the probability was low. This agrees with the findings of Hutt and Greenwood (1929) and of Byerly, Titus and Ellis (1933a and b). The latter named authors (1933a) attributed the variable incidence of chondrodystrophy as due to some deficiency of the ration yet this would hardly account for the differences noted in the present study since all birds received the same ration. Breeding would seem to be a more logical explanation of the variability.

The incidence of chondrodystrophy in fifty-three individual matings that produced this character are given in table 13 together with the percentage of embryos affected and ratios approached, that is, the proportion of normal embryos to chondrodystrophic embryos. The fifteen sires included in this table were selected because of the relatively high incidence of chondrodystrophy in their progeny. All dams to which they were mated are included in the table, therefore complete families of progeny are represented. The sires producing a larger number of chondrodystrophic embryos were used because such matings include the dams that produced the lowest ratios of normal to chondrodystrophic embryos, they also include many hens that produce no chondrodystrophy, and others that produce wide ratios. In other words, all types of ratios that were observed are represented in table 13. For the data in the first half of table 13 the number of embryos alive on the fifth day was used as a basis in obtaining the ratios, while for the other data the number of total fertile eggs set was used. The number of embryos alive on the fifth day was not available for the latter material. The results of a limited number of matings are summarized in table 14.

It is much to be regretted that the 1931 matings which produced the higher incidences of chondrodystrophy could not be repeated. Few progeny were available from the 1932 matings which produced high percentages of this characteristic. As a consequence of these circumstances no critical test of inheritance could be made, however the meager breeding data available are presented. The data of tables 13 and 14 are of interest in that they suggest that if chondrodystrophy is inherited it is recessive in character with at least three pairs of

TABLE 13

Distribution of Chondrodystrophy By Dams—For Fifteen Sires With High Incidence of This Character.

Sire's No.	Dam No.	No. of 5th Day Living Embryos	Incidence of Chondrodystrophy		Ratios Approached[1]
			No.	Percent	
97	253	49	1	2.04	63 :1
	2407	8	2	25.00	3 :1 or 7 :1
	2446	20	3	15.00	7 :1 or 15 :1
	2478	50	1	2.00	63 :1
	2563	33	3	9.09	7 :1 or 15 :1
Totals for producer dams..		160	9	.....	.....
Also 1 negative mate.					
155	4256	59	6	10.17	15 :1
	4257	17	3	17.65	13 :3 or 3 :1
	4278	31	4	12.90	7 :1
	4280	38	10	26.32	3 :1
Totals for producer dams..		145	23	.....	.....
Also 2 negative mates.					
456	4779	70	2	2.86	63 :1
	4788	75	1	1.33	63 :1
Totals for producer dams..		145	3	.....	.....
Also 2 negative mates.					
3261	4858	74	6	8.11	15 :1
	4865	102	1	.98	63 :1
	4867	38	2	5.26	15 :1
	4870	55	2	3.64	15 :1
	4871	41	2	4.88	15 :1
Totals for producer dams..		310	13	.....	.....
Also 9 negative mates.					
3719	4728	37	3	8.11	15 :1
Also 3 negative mates.					

[1] The most closely approached common ratios are designated. All ratios suggested are within the range of .95 and .05. The 7 :1 ratios would be obtained in backcross matings.

TABLE 13—Continued.

Distribution of Chondrodystrophy By Dams—For Fifteen Sires With High Incidence of This Character.

Sire's No.	Dam No.	No. of 5th Day Living Embryos	Incidence of Chondrodystrophy		Ratios Approached[1]
			No.	Percent	
4016	4734	58	2	3.45	63:1
	4738	87	4	4.60	15:1
	4740	68	1	1.47	63:1
	4741	48	1	2.08	63:1
	4747	48	5	10.42	7:1 or 15:1
Totals for producer dams.. Also 1 negative mate		309	13	.....	.....
4790	3865	27	3	11.11	7:1 or 15:1
	3910	33	1	3.03	63:1
	4753	69	3	4.35	15:1
Totals for producer dams.. Also 1 negative mate		129	7	.....	.....
8118	4795	18	4	22.22	3:1
	4797	38	1	2.63	63:1
	4798	16	3	18.75	3:1 or 15:1
	4799	29	1	3.45	15:1
Totals for producer dams.. Also 4 negative mates		101	9	.....	.....
2533	3106	38	2	5.26	15:1
	3121	33	14	42.42	3:1
Totals for producer dams.. Also 11 negative mates		71	16	.....	.....
3214	3226	43	12	27.91	3:1
	3227	37	1	2.70	63:1
	3327	21	1	4.76	15:1
Totals for producer dams.. Also 8 negative mates		101	14	.....	.....

[1] The most closely approached common ratios are designated. All ratios suggested are within the range of .95 and .05. The 7:1 ratios would be obtained in backcross matings.

TABLE 13—Continued.

Distribution of Chondrodystrophy By Dams—For Fifteen Sires With High Incidence of This Character.

Sire's No.	Dam No.	No. of 5th Day Living Embryos	Incidence of Chondrodystrophy		Ratios Approached[1]
			No.	Percent	
4962	3126	16	1	6.25	15:1
	3143	22	3	13.64	7:1
	3303	14	2	14.29	7:1
	3333	25	1	4.00	15:1
	3341	20	3	15.00	7:1
	3353	28	1	3.57	15:1
Totals for producer dams..		125	11	.....	.....
Also 9 negative mates					
135	4	26	1	3.85	15:1
	272	24	1	4.17	15:1
	309	22	1	4.54	15:1
	346	42	2	4.76	15:1
	380	18	2	11.11	7:1
	2470	18	2	11.11	7:1
	2483	38	1	2.63	63:1
Totals for producer dams..		188	10	.....	.....
Also 8 negative mates					
112	226	42	1	2.38	63:1
	305	52	4	7.69	15:1
	2494	59	1	1.69	63:1
Totals for producer dams..		153	6	.....	.....
Also 10 negative mates					
126	335	48	4	8.33	15:1
Also 15 negative mates					
204	12	32	1	3.12	63:1
	236	48	4	8.33	15:1
Totals for producer dams..		80	5	.....	.....
Also 13 negative mates					

[1] The most closely approached common ratios are designated. All ratios suggested are within the range of .95 and .05. The 7:1 ratios would be obtained in backcross matings.

TABLE 14

Breeding Results Concerning Chondrodystrophy

Sire's Parents		Ratio of chondrodystrophy in progeny of parents	Sire	Sire mated to[1]	Ratios of chondrodystrophy approached		
Sire	Dam				With full sisters	With half-sisters	With outcross females[2]
1518	3331	Negative mating[4]	135	One full sister and 14 half-sisters	1—Negative mating	7—Negative matings 2— 7:1 ratios 4—15:1 ratios 1—63:1 ratio	
2533	3400	Negative mating	112	Five full sisters and two half-sisters	3—Negative matings 1—15:1 ratio 1—63:1 ratio	1—Negative mating	
3119	3145	Negative mating	204	Two full sisters, two half-sisters and two outcross females	2—Negative matings	1—Negative mating 1—15:1 ratio	1—Negative mating 1—63:1 ratio
1467	3385	Negative mating	126	Three full sisters and four outcross females	3—Negative matings		3—Negative matings 1—15:1 ratio
2533	3106	15:1 ratio	97	Four half-sisters and one outcross female		2— 7:1 ratios 2—63:1 ratios	1— 3:1 ratio[3]
				Summary	9—Negative matings 1—15:1 ratio 1—63:1 ratio	9—Negative matings 4— 7:1 ratios 5—15:1 ratios 4—63:1 ratios	4—Negative matings 1— 3:1 ratio 1—15:1 ratio 1—63:1 ratio
				Grand Totals		22—Negative matings 1— 3:1 ratio 4— 7:1 ratios 7—15:1 ratios 6—63:1 ratios	

[1] The females to which the sire was mated were also from negative matings with the one exception noted.

[2] Outcross—outcrossed only in the sense of no inbreeding in this generation although from same inbred strain.

[3] This female was also from a mating producing a 3:1 ratio.

[4] A negative mating is a mating from which no chondrodystrophic embryos were obtained.

factors involved. The ratios of thirteen additional dams mated in 1933 to several other males are quite similar to those given. Landauer and Dunn (1926), Hutt and Greenwood (1929) and Munro (1932) have similarly expressed the belief that chondrodystrophy has a complex hereditary basis, while Dunn (1927) and Byerly, Titus and Ellis (1933 a and b) have concluded that it is not inherited. Further planned matings must be made before final conclusions are drawn.

MALPOSITIONS

Incidence of Malpositions—Year and Institution

The incidence of malpositions according to year and institution is given in table 15. Description of the several malpositions and the numbers by which they are designated in this study are given in this table. In later tables the malpositions are identified by number only.

The purpose in presenting the data in table 15 is to illustrate the great variation encountered in the occurrence of malpositions in different years and at the two different institutions. Certain malpositions varied widely from group to group as did the frequency to total malpositions.

Incidence of Malpositions—Date Eggs Were Set

The several malpositions were present somewhat irregularly for the different setting dates (table 16) but no definite seasonal trends are noted in any case except that in the Iowa (1933) group malposition 9 (feet over head) occurred much more frequently in the January settings. This single irreguarity is probaby accidental since no consistent trend is present. Total malpositions also failed to reveal any general seasonal trend although for the four combined groups the incidence was somewhat less for the settings in May, September-October, and November-December, and those for the latter part of January and for February.

Incidence of Malpositions—Character of Mating

The frequency with which malpositions occurred in different types of matings is shown in table 17. The data given are based upon total dead embryos throughout the entire period of incubation and upon the number of embryos that were living on the 18th day. It may be noted that the occurrence of malpositions varies widely for the different groups. Attention is called in particular to the increased frequency of total malpositions which is based upon 18th day living embryos in the highly inbred matings, both parents dwarfism carrier matings, creeper matings and Albino Wyandotte matings.

TABLE 15

Incidence of Various Malpositions

By Year and Institution

Institution	Year	No. fertile eggs set	No. 18th day living embryos	Malpositions									Total No. malpositions	Percent malpositions of 18th day living embryos
				Head between thighs 1	Head in small end of egg 2	Head to left 3	Rotated in shell 4	Head not under wing 5	Positions 3 and 5 combined 6	Positions 4 and 5 combined 7	Positions 1 and 2 combined 8	Feet over head 9		
				Percent of 18th day living embryos										
Iowa	1931	1185	864	6.02	1.97	1.62	2.43	.35	.23	0.00	.23	.12	112	12.97
Louisiana	1931	900	747	1.20	2.68	.67	1.07	.27	0.00	0.00	0.00	.13	45	6.02
Louisiana	1932	2943	2511	.48	1.43	.48	1.00	.16	.04	0.00	.04	0.00	91	3.63
Louisiana	1933	4082	3359	1.28	2.89	.83	1.37	2.50	.06	.45	.24	0.00	323	9.62
Iowa	1932	2733	2277	1.10	1.45	2.99	.53	4.08	.04	.88	.31	0.00	259	11.38
Iowa	1933	4619	4149	.99	2.63	3.57	.79	10.07	.05	.12	.17	1.40	821	19.79

TABLE 16

Incidence of Malpositions

By Date Eggs Were Set—Percent of Total Dead Embryos

Date eggs were set	Malpositions						Total malpositions	Total No. dead
	1	2	3	4	5	9		
Four groups combined[1]								
January 1-17	4.62	13.85	1.54	10.77	4.62	.....	35.40	65
January 18-30 ...	2.42	7.27	.61	7.88	3.03	.....	21.21	165
February 1-16 ...	3.46	6.05	2.59	4.32	4.61	.....	21.03	347
February 18-27 ..	4.33	5.51	5.12	1.97	2.76	.....	19.69	254
March 1-13	7.19	6.83	3.96	1.80	6.12	.....	25.90	278
March 18-30	8.66	8.66	3.54	4.72	4.33	.....	29.91	254
April 1-14	9.21	8.33	4.39	6.58	8.33	.44	37.28	228
April 15-29	5.97	9.70	1.49	6.72	2.24	.....	26.12	268
May (including 1 hatch in June) .	4.35	4.78	.43	3.04	4.78	.43	17.81	230
Sept.-Oct.	3.47	6.44	1.49	2.97	.99	.....	15.36	202
Nov.-Dec.	1.52	6.60	1.02	5.58	6.09	.....	20.81	197
Total number examined	129	179	64	114	109	2	597	2488
Iowa 1933								
January 9, 16....	4.60	3.45	12.64	1.15	24.14	25.29	71.27	87
January 23, 30 ...	4.06	8.12	9.14	2.54	16.24	19.29	59.39	197
February 6, 13 ...	3.91	10.16	7.81	.78	35.94	0.00	58.60	128
February 20, 27 ..	4.50	7.21	7.21	3.60	21.62	0.00	44.14	111
March 6, 13	1.58	6.62	8.52	2.21	25.24	.63	44.80	317
March 20, 27	1.97	7.30	8.43	2.25	28.09	.28	48.32	356
April 3, 10	2.33	6.41	11.08	3.50	35.86	.29	59.47	343
Number for total examination ...	42	109	142	38	426	64	821	1539
Percent for total examination ...	2.73	7.08	9.23	2.47	27.68	4.16	53.35	

[1] Iowa 1931, Louisiana 1931, 1932 and 1933.

TABLE 17

Incidence of Various Malpositions

By Character of Mating

Character of mating		Total number dead embryos	Number fertile eggs set	Malpositions						Total number	Total percent
				1	2	3	4	5	9		
Regular 0	Percent of dead embryos	1458	5521	5.08	6.17	2.67	4.46	1.85	.14	297	20.37
	Percent of 18th day living	.....	.....	1.58	1.92	.83	1.39	.58	.04	.....	6.34
Mildly inbred 1	Percent of dead embryos	211	983	6.64	8.53	1.42	9.95	10.43	.....	78	36.97
	Percent of 18th day living	.....	.....	1.55	2.00	.33	2.33	2.44	.....	.....	8.65
Highly inbred-malposition stock 2	Percent of dead embryos	2429	7352	3.29	6.42	9.02	2.88	22.19	2.39	1122	46.19
	Percent of 18th day living	.....	.....	1.24	2.43	3.41	1.09	8.39	.90	.....	17.46
Sire dwarf carrier 3	Percent of dead embryos	132	481	4.55	4.55	1.52	2.27	13.64	.....	35	26.53
	Percent of 18th day living	.....	.....	1.51	1.51	.51	.75	4.52	.....	.....	8.80
Both parents dwarf carriers 4	Percent of dead embryos	289	794	4.50	8.65	1.73	4.84	10.03	.....	86	29.75
	Percent of 18th day living	.....	.....	1.94	3.73	.75	2.09	4.32	.....	.....	12.83
Both parents creepers 9	Percent of dead embryos	55	84	1.82	7.27	1.82	5.45	5.45	.....	12	21.81
	Percent of 18th day living	.....	.....	1.67	6.67	1.67	5.00	5.00	.....	.....	20.01
Kiwi mating 10	Percent of dead embryos	45	222	11.11	17.78	0.00	2.22	4.44	.....	16	35.55
	Percent of 18th day living	.....	.....	2.44	3.90	0.00	.49	.98	.....	.....	7.81
Albino Wyandotte mating 11	Percent of dead embryos	94	180	5.32	8.51	4.26	1.06	5.32	.....	23	24.47
	Percent of 18th day living	.....	.....	3.73	5.97	2.99	.75	3.73	.....	.....	17.17
Sire sticky carrier and both parents dwarf carriers 15	Percent of dead embryos	115	225	6.09	13.04	3.48	3.48	5.22	.....	36	31.31
	Percent of 18th day living	.....	.....	3.95	8.47	2.26	2.26	3.39	.....	.....	20.33
Miscellaneous matings	Percent of dead embryos	89	620	.....	.....	.....	.....	.....	.....	19	21.35
Averages—All matings except highly inbred malposition	Percent of dead embryos	2488	9110	5.10	7.32	2.57	4.62	4.54	.08	602	24.20
	Percent of 18th day living	.....	.....	1.71	2.44	.86	1.55	1.52	.03	.....	8.12

Incidence of Malpositions—Sires

The number of specific types of malpositions is given in tables 9 and 10 for each sire together with the total number of malpositions and the percent total malpositions of 18th day living embryos. Attention is called to the fact that in every case in which the number of malpositions for each sire is appreciable, all five of the common types of malpositions are represented. Malposition 9 (feet over head) was rarely found except in the Iowa (1933) examinations, here six sires of the ten used produced malpositions of this type.

Incidence of Malpositions—Individual Matings and By Sexes

Table 18 presents the results of tests for homogeneity of the occurrence of malpositions in 309 individual matings involving 8,562 embryos alive on the 18th day. For the entire sample involved it is revealed that real variation in the occurrence of malpositions existed from dam to dam. This also holds true for the different dams mated to a given sire in 20 of 29 cases. If the incidence of malpositions were due to chance variation alone we might expect greater homogeneity, but if dependent upon some cause, such as inherent differences, some subsamples might well be homogeneous, others heterogeneous.

Tables 19, 20 and 21 present evidence as to the incidence of malpositions by sexes. Table 19 gives the summarized results for 701 male embryos, 762 females and 1,025 of unidentified sex for data in which all dead embryos were examined. The sexes of the progeny showing malpositions, from seventeen males producing a high incidence of these abnormalities, are revealed in table 20. In table 21 is presented a typical example of the distribution of malpositions by sexes for the various females mated to one male. Distribution of malpositions are quite similar for the two sexes, both on a basis of percentage of each sex that died and in actual numbers for the Louisiana data (table 19). For the data by sires, given in table 20, the incidences of malpositions in the two sexes are similar when considered on a basis of percentage of each sex that died but not in actual number of malpositions. The percentage distributions of the respective malpositions for all sires combined are remarkably similar for both sexes. However, attention is called to the fact that the inbred sires, table 20, produced 608 female malpositions as compared to 353 male malpositions. In other words, 63.27 percent of all malpositions produced by these sires were females. It may be noted also that 61.75 percent of all dead embryos sexed were females, with great variation for the different sires. These facts are considered in connection with discussion of a sex-linked lethal gene.

It is interesting to note, in table 20, that for either sex approximately 4 percent of all sexed embryos were in position 1 (head between thighs); 8 percent in position 2 (head in small end of egg); 13

TABLE 18

Test for Homogeneity of Occurrence of Malpositions

Individual Matings

Sire	Number of dams	Degrees of freedom	Number 18th day living embryos	Number of malpositions	Percent malpositions	Chi-square with significance[1]
97	6	5	147	23	15.65	30.010†
155	6	5	141	19	13.48	6.664
456	11	10	305	27	8.85	31.736†
3261	14	13	492	66	13.42	33.235†
3719	4	3	100	13	13.00	28.588†
4016	6	5	267	45	16.85	5.107
4597	9	8	216	28	12.96	10.123
4740	3	2	47	9	19.15	1.155
4790	4	3	173	12	6.94	2.097
8118	10	9	176	33	18.75	20.672*
1727	9	8	255	37	14.51	11.257
1467	13	12	239	31	12.97	42.566†
1518	12	11	264	28	10.61	23.095*
2505	7	6	90	7	7.78	7.236
5124	5	4	117	14	11.97	16.004†
438	10	9	273	7	2.56	14.277
2533	13	12	337	77	22.85	69.965†
3119	10	9	345	31	8.99	32.067†
3191	13	12	438	16	3.65	27.442†
3214	10	9	163	22	13.50	43.565†
4962	15	14	270	39	14.44	20.918
105	14	13	475	67	14.10	86.816†
112	11	10	376	75	19.95	32.476†
135	15	14	394	103	32.42	32.424†
170	19	18	713	78	10.94	55.453†
202	17	16	500	141	28.20	46.534†
204	15	14	488	85	17.42	86.450†
3172	13	12	459	84	18.30	34.062†
126	15	14	302	111	36.78	81.187†
Totals	309	280	8,562	1,328	15.51	933.181†

[1] * P of between .02 and .01.
† P of less than .01.

TABLE 19

Incidence of Malpositions According to Sex of Embryo

Data of Iowa '31, Louisiana '31, '32 and '33 Summarized

Normal dead	Malpositions									Total number malpositions	Total number dead	Percent classified as malpositions
	1	2	3	4	5	6	7	8	9			
Males (Malpositions as percent of total dead males)												
62.62	8.13	10.27	4.42	6.70	5.14	.29	.71	1.43	.29	262	701	37.38
Females (Malpositions as percent of total dead females)												
62.99	6.56	12.07	3.15	6.43	6.96	.39	1.31	.13	0.00	282	752	37.01
Sex Unknown (Malpositions as percent of total dead of unknown sex)												
97.37	.88	.59	.39	.39	.39	0.00	0.00	0.00	0.00	27	1025	2.63[1]

[1] Incidence of malpositions is negligible in this group because most of the embryos so classified died early in the incubation period.

TABLE 20

Incidence of Malpositions According to Sex of Embryos

By Sires (Highly Inbred Leghorns)

Iowa 1932 Sire	No. 18th Day Living Embryos	Normal Dead Males	Male Malpositions (As percent of total male dead)									Total No. Male Malpositions	Total No. Dead Males
			1	2	3	4	5	6	7	8	9		
B438	265	37.50	0.00	12.50	25.00	0.00	12.50	0.00	0.00	12.50	0.00	5	8
B1467	239	54.84	3.23	9.68	3.23	3.23	25.81	0.00	0.00	0.00	0.00	14	31
B1518	264	14.29	14.29	0.00	35.71	0.00	35.71	0.00	0.00	0.00	0.00	12	14
B2533	337	44.44	5.56	3.70	24.07	0.00	20.37	0.00	1.85	0.00	0.00	30	54
B3119	354	28.57	7.14	14.29	0.00	7.14	35.71	0.00	7.14	0.00	0.00	10	14
B3191	438	46.15	23.08	0.00	7.69	7.69	15.38	0.00	0.00	0.00	0.00	7	13
B3214	175	43.75	12.50	6.25	25.00	6.25	6.25	0.00	0.00	0.00	0.00	9	16
B4962	270	35.29	0.00	11.76	11.76	0.00	29.41	0.00	11.76	0.00	0.00	11	17
Iowa 1933													
C105L	475	38.46	0.00	10.26	12.82	0.00	33.33	0.00	0.00	0.00	5.13	24	39
C111L	214	50.00	0.00	6.25	18.75	6.25	18.75	0.00	0.00	0.00	0.00	8	16
C112E	376	32.00	2.00	6.00	10.00	4.00	34.00	0.00	0.00	2.00	10.00	34	50
C126B	333	20.83	8.33	12.50	8.33	2.08	37.50	0.00	0.00	0.00	10.42	38	48
C135B	394	44.68	6.38	6.38	4.26	4.26	34.04	0.00	0.00	0.00	0.00	26	47
C170I	713	35.90	0.00	7.69	2.56	2.56	51.28	0.00	0.00	0.00	0.00	25	39
C202E	503	38.10	0.00	6.35	11.11	3.17	33.33	0.00	0.00	0.00	7.94	39	63
C204I	488	34.04	2.13	14.89	14.89	4.26	25.53	2.13	2.13	0.00	0.00	31	47
B3172	463	28.57	2.38	7.14	19.05	7.14	35.71	0.00	0.00	0.00	0.00	30	42
Grand Totals													
Number	6,301	205	22	45	70	18	173	1	5	2	17	353	558
Percent ..... (All Iowa '32 and Iowa '33 Sires)	.....	36.74	3.94	8.06	12.54	3.23	31.00	0.18	0.90	0.36	3.05	.....	.....

TABLE 20 (Continued)

Incidence of Malpositions According to Sex of Embryos

By Sires (Highly Inbred Leghorns)

Percent Males Classified as Malpositions	Normal Dead Females	Female Malpositions (As percent of total female dead)									Total No. Female Malpositions	Total No. Females Dead	Percent Females Classified as Malpositions	No. Sex Unknown
		1	2	3	4	5	6	7	8	9				
62.50	0.00	0.00	50.00	25.00	25.00	0.00	0.00	0.00	0.00	0.00	4	4	100.00	0
45.16	51.28	5.13	7.69	0.00	0.00	33.33	0.00	0.00	2.56	0.00	19	39	48.72	2
85.71	36.00	8.00	12.00	20.00	0.00	12.00	0.00	8.00	4.00	0.00	16	25	64.00	3
55.56	44.05	2.38	4.76	13.10	3.57	25.00	0.00	7.14	0.00	0.00	47	84	55.95	10
71.43	26.67	10.00	13.33	3.33	6.67	13.33	0.00	16.67	10.00	0.00	22	30	73.33	1
53.85	67.86	0.00	0.00	10.71	3.57	10.71	0.00	3.57	3.57	0.00	9	28	32.14	2
56.25	26.09	8.70	17.39	26.09	4.35	13.04	0.00	4.35	0.00	0.00	17	23	73.91	7
64.71	30.55	5.56	5.56	30.55	2.78	16.67	2.78	5.56	0.00	0.00	25	36	69.44	10
61.54	22.22	3.10	11.11	12.96	1.85	38.89	0.00	1.85	0.00	7.41	42	54	77.78	6
50.00	38.89	5.56	11.11	11.11	11.11	22.22	0.00	0.00	0.00	0.00	11	18	61.11	12
68.00	36.36	3.64	5.45	12.73	1.82	32.73	0.00	0.00	0.00	7.27	35	55	63.64	10
79.17	20.43	4.30	10.75	12.90	1.08	37.63	0.00	0.00	0.00	12.90	74	93	79.57	17
55.32	19.75	14.81	7.41	12.35	6.17	32.10	0.00	1.23	0.00	6.17	65	81	80.25	19
64.10	24.19	1.61	19.35	12.90	3.23	38.71	0.00	0.00	0.00	0.00	47	62	75.81	12
61.90	32.77	1.68	4.20	8.40	0.00	38.66	0.00	0.00	0.00	14.29	80	119	67.23	26
65.96	32.39	7.04	11.27	18.31	1.41	23.94	1.41	0.00	1.41	2.82	48	71	67.61	16
71.43	40.51	0.00	5.06	12.66	2.53	34.18	0.00	2.53	0.00	2.53	47	79	59.49	20
.....	293	42	78	117	24	271	2	21	7	46	608	901	.....	173
63.26	32.52	4.66	8.66	12.99	2.66	30.08	0.22	2.33	0.78	5.11	.....	.....	67.48	2.74[1]

[1] Of total 18th day living embryos.

TABLE 21

Incidence of Malpositions According to Sex of Embryos

By Individual Matings—A typical Example

Sire No.	Dam No.	No. of 18th day living	No. of Normal Dead Males	Male Malpositions (Number of)								
				1	2	3	4	5	6	7	8	9
C202E	C 54	1	0	0	0	0	0	0	0	0	0	0
C202E	C 58	37	2	0	0	1	1	2	0	0	0	2
C202E	C 60	45	4	0	1	1	0	2	0	0	0	0
C202E	C 79	17	0	0	1	0	0	0	0	0	0	0
C202E	C 110	45	0	0	1	0	0	2	0	0	0	0
C202E	C 119	11	0	0	0	0	0	3	0	0	0	0
C202E	C 130	39	0	0	1	1	0	0	0	0	0	2
C202E	C 137	38	5	0	0	1	1	4	0	0	0	0
C202E	C 162	49	2	0	0	0	0	3	0	0	0	0
C202E	C 204	22	1	0	0	0	0	3	0	0	0	0
C202E	C 250	10	0	0	0	0	0	0	0	0	0	0
C202E	C 271	33	1	0	0	0	0	0	0	0	0	0
C202E	C 285	19	0	0	0	0	0	0	0	0	0	1
C202E	C 311	17	1	0	0	3	0	0	0	0	0	0
C202E	C2510	22	1	0	0	0	0	0	0	0	0	0
C202E	C2513	42	1	0	0	0	0	0	0	0	0	0
C202E	C2579	56	6	0	0	0	0	2	0	0	0	0
Total Number		503	24	0	4	7	2	21	0	0	0	5
Total Percent		...	38.10	0.00	6.35	11.11	3.17	33.33	0.00	0.00	0.00	7.94

TABLE 21 (Continued)

Incidence of Malpositions According to Sex of Embryos

By Individual Matings—A typical Example

Total No. Male Malpositions	Total No. Males Dead	No. of Normal Dead Females	Female Malpositions (Number of)									Total No. Female Malpositions	Total No. Females Dead	No. Dead Sex Unknown
			1	2	3	4	5	6	7	8	9			
0	0	0	0	0	0	0	1	0	0	0	0	1	1	0
6	8	6	0	0	0	0	2	0	0	0	1	3	9	2
4	8	3	0	0	2	0	0	0	0	0	1	3	6	4
1	1	0	0	0	0	0	0	0	0	0	3	3	3	1
3	3	4	0	0	2	0	6	0	0	0	0	8	12	2
3	3	0	0	0	0	0	1	0	0	0	0	1	1	0
4	4	1	0	0	1	0	4	0	0	0	1	6	7	0
6	11	3	0	0	1	0	7	0	0	0	2	10	13	1
3	5	4	1	0	0	0	2	0	0	0	2	5	9	3
3	4	2	0	2	1	0	3	0	0	0	0	6	8	3
0	0	0	0	1	0	0	0	0	0	0	0	1	1	0
0	1	0	0	1	0	0	3	0	0	0	0	4	4	1
1	1	3	0	0	0	0	4	0	0	0	0	4	7	1
3	4	0	1	0	3	0	5	0	0	0	1	10	10	3
0	1	7	0	0	0	0	3	0	0	0	0	3	10	0
0	1	4	0	1	0	0	3	0	0	0	4	8	12	3
2	8	2	0	0	0	0	2	0	0	0	2	4	6	2
39	63	39	2	5	10	0	46	0	0	0	17	80	119	26
61.90		32.77	1.68	4.20	8.40	0.00	38.66	0.00	0.00	0.00	14.29	67.23		.....

percent in position 3 (head to left); 3 percent in position 4 (rotated in shell); 30 percent in position 5 (head not under wing) and 3 percent of dead males and 5 percent of dead females were in position 9 (feet over head). In a few instances a given embryo was found in two different malpositions, a combination of positions four and five being most frequent.

The relative frequency of the different malpositions for different stock is of interest. For the data of the non-inbred stock position 2 (head in small end of egg) was most commonly found. This agrees with the results of Smith (1931) and Byerly and Olsen (1931). In the work of Hutt (1929) positions 2 and 4 occurred with about equal frequency and more often than did the other positions. Position 1 (head between legs), position 4 (rotated) and position 5 (head not under wing) were found at approximately the same rate in the non-inbred stock. In the highly inbred stock, in which the incidence of malpositions was much higher, position 5 (head not under wing) occurred much more frequently than did any other position. This is in agreement with report of Hutt and Pilkey (1934). This position was not included in the studies of earlier workers. Position 3 (head to left) ranked second in occurrences in the highly inbred stock of this study as in the data of Hutt and Pilkey (1934), with position 1 next in order. The other positions occurred with about equal frequency.

It has been observed, as may be noted in table 21, that often the dam produces two or three, and in some instances more, different types of malpositions. This fact, together with similar observations concerning sires (table 20 and previous tables), suggests that possibly no innate differences underlie specific malpositions. If this be true an impaired ability of orientation may be the basic underlying factor for all malpositions.

The variation in incidence of the different positions in different stock as reported by previous investigators might be interpreted to indicate genetic differences for each position. However, all workers have found several positions in their material and it seems unlikely that in stock from various sources all would carry the several multiple recessive genes required if each position were inherited separately. It is definitely indicated that no one position is inherited as a simple recessive.

The suggestion is offered that an impaired sense of orientation is the basic condition involved and that the several specific positions are merely different expressions of the same trait. It is freely admitted that the data available do not prove the hypothesis suggested. It will be necessary to make extensive planned matings of stock of known malposition breeding before conclusive evidence is available. Extensive tests are also necessary to determine the number of malpositions of various sorts that can be induced to occur by various

environmental influences, in a stock standardized as to incidence of malpositions.

Another essential point that needs to be definitely determined is the absolute degree to which each respective position is lethal. Some of the positions are believed to be an absolute bar to hatching, for example head between thighs; while it is known that some chicks in other malpositions do hatch, for example embryos with the head in the small end of the egg. No comprehensive test has been made to determine the percent of each position that does hatch.

Sex Ratios

Sex was determined for the dead embryos by macroscopic examination of the gonads, when possible to do so. A small number of older embryos were not sexed due to decomposition of embryos or to accidents of examination, but most of the embryos that died after 12 days of incubation and a number that died before this time were sexed. Tables 22 and 23 give the number of dead embryos of undetermined sex, the number of males, and number of females and the significance of the deviation from a 1:1 ratio for the number of males and females identified. The proportion of the two sexes is given by date eggs were set in table 22.

Consideration of the sex ratio for the combined groups by date eggs were set shows no significant differences, except possibly for the March 18-30 settings. Embryos of the Iowa (1933) group yielded a consistently significant excess of females for all dates upon which eggs were set. Since this tendency was constant, date of setting eggs did not affect the sex ratio. It may be stated that the sex ratio did not vary sensibly according to season. This is contrary to early work of Jull (1924) but is in agreement with the results of Lambert and Knox (1926).

Sex ratios for embryos grouped according to breeds and according to character of mating are given in table 23.

The only significant deviation of the sex ratio when considered by breeds is in the case of the miscellaneous breeds. Analysis of the data for this group, by sires, failed to indicate a sex-linked lethal gene. It should be noted that the highly inbred single comb White Leghorns are not included in the White Leghorn breed as given.

The highly inbred Leghorn group is the only type of mating which shows an abnormal sex ratio. Further analysis is made of this group.

Significant and striking deviations from the normal sex ratio as reported by the extensive work of Pearl (1917) and other workers listed by Jull (1932) were obtained in the highly inbred group.

That no differential sex mortality of embryos occurs normally has been determined conclusively by Thomsen (1911), Pearl (1917),

TABLE 22

Sex Ratios of Dead Embryos

By Date Eggs Were Set

Date eggs were set	Sex unknown	Number males	Number females	Sex ratio with significance of Chi-square[1]
		Four groups combined[2]		
January 1-17	25	21	19	52.50
January 18-30	49	56	60	48.28
February 1-16	127	111	109	50.45
February 18-27	112	71	71	50.00
March 1-13	119	85	74	53.46
March 18-30	81	70	103	40.46*
April 1-14	81	72	75	48.98
April 15-29	116	78	74	51.32
May	132	48	50	48.98
Sept.-Oct.	90	46	66	41.07
Nov.-Dec.	95	42	60	41.18
Totals	1027	700	761	47.91
		Iowa 1933		
January 9, 16	10	28	49	36.37†
January 23, 30	50	53	94	36.06†
February 6, 13	26	40	62	39.22*
February 20, 27	20	26	65	28.57†
March 6, 13	88	76	153	33.19†
March 20, 27	74	111	171	39.36†
April 3, 10	196	72	75	48.98
Totals	464	406	669	37.77†

[1] If Chi-square is not significant no indicator is used.

* — P of between .05 and .01, Chi-square table p. 96, Fisher (1930).

† — Highly significant difference, i. e., P less than .01.

[2] Iowa 1931, Louisiana 1931, Louisiana 1932 and Louisiana 1933.

TABLE 23

Sex Ratios of Dead Embryos

By Breeds and by Character of Mating

Breeds	Number unknown sex	Number males	Number females	Sex ratio with significance of Chi-square[1]	Character of mating	Number unknown sex	Number males	Number females	Sex ratio with significance of Chi-square[1]
Miscellaneous breeds and crossbreds	73	83	120	40.89†	Regular	664	370	424	46.60
White Leghorn	267	172	154	52.76	Mildly inbred	66	73	72	50.34
R. I. Red	423	200	218	47.85	Highly inbred Leghorns	855	573	939	37.90†
White Wyandotte	120	103	115	47.25	Sire dwarf carrier	71	32	29	52.46
Red by Leghorn crossbred	86	78	92	45.88	Both sire and dam dwarf carriers	109	86	94	47.78
Red by sticky crossbred	56	65	63	50.78	Both sire and dam sticky carriers	15	11	13	45.83
					Both sire and dam creepers	14	19	22	46.34
					Kiwi mating	18	13	14	48.15
					Albino Wyandotte	27	38	29	56.72
					Sire sticky carrier and both parents dwarf carriers	26	45	44	50.56

[1] † Highly significant difference, i. e., P. less than .01.

Jull (1924), Lambert and Knox (1926), Lambert and Curtis (1929) and Horn (1927). The same is true of this study except for the highly inbred White Leghorn matings.

Sex Ratios—Sires

Sex ratios by sires are given in tables 9 and 10 for all sires that fertilized as many as 100 eggs. In no case did a sire included in table 9 show a conclusively large excess of either sex although five borderline ratios were obtained. However these all involved relatively small numbers. In only one case, of the 5 mentioned, was there a possible excess of males found.

On the other hand, of the known sixteen highly inbred single comb White Leghorn males (table 10) five produced a highly significant excess of females and six other ratios are of borderline significance. These results suggested the presence of a sex-linked lethal gene and further analyses were undertaken to test this possibility.

A Sex-Linked Lethal Factor

In table 24 is presented breeding evidence indicating the presence of a sex-linked lethal gene. The sex ratios are given of the progeny of certain sires and the sex ratios of the progeny of their sons that were used the following season. The sex ratios of the malpositions are given for the same males.

Because the female is heterozygous for sex in the domestic fowl the sire is the parent that transmits a sex-linked lethal factor. Males with the genotype ZZ Ll mated to females (WZL)—would produce living progeny with a sex ratio of two males to one female because one-half of the females would not hatch since they would receive the lethal gene from their sire. Conversely such a mating would be expected to produce a sex ratio of dead embryos of two females to one male, or, as generally expressed, a sex ratio of 33.33. One half of the males from this type of mating would be carriers of the lethal gene (ZZLl) but would be viable.

The results presented in table 24 strongly support the theory that a sex-linked lethal gene was carried by certain males used in 1932, namely; 1518, 3119, and 1467 and that it was transmitted by them to their sons used the following season. Sire 2533 probably likewise carried the lethal factor even though his two sons used in 1933 proved to be non-carriers. As stated above, only one-half of the sons would be expected to transmit the factor. It is probable that sires 3191 and 4962 were also carriers of the lethal factor although no sons from these males were used the following season. It may be noted that the number of observed females that died is in some cases appreciably less than the number expected but it should be mentioned that not all

TABLE 24

Breeding Evidence of a Sex-Linked Lethal Factor

Sex ratios of dead embryos

Band number	Number dead females expected	1932 Sires: No. of each sex observed		Sex ratio with significance of Chi-square[1]	1932 Sires: No. of each sex of malposition		Sex ratio of malposition	Band number	Number dead females expected	Carrier sons of sires used in 1932: No. of each sex observed		Sex ratio with significance of Chi-square	Carrier sons of sires used in 1932: No. of each sex of malposition		Sex ratio of malposition	Band number
		Males	Females		Males	Females				Males	Females		Males	Females		
1518	73	14	25	35.90*	12	16	42.86	135	110	47	81	36.43†	26	65	28.57	.....
2533	109	54	84	39.13*	30	47	38.96	.....	.....	.....	.....	.....	.....	.....	.....	105
3119	95	14	30	31.82*	10	22	31.25	202	137	63	119	34.43†	39	80	32.77	112
								204	133	47	71	39.83†	31	48	39.24	.....
3191	?	13	28	31.71*	7	9	43.75	No sons used.....		.....	.....	.....	.....	.....	.....	.....
4962	88	17	36	32.14†	11	25	29.73	No sons used.....		.....	.....	.....	.....	.....	.....	.....
1467	78	14	19	44.29[2]	14	19	42.42	126	100	48	93	34.04†	38	74	33.93	.....
438	?	8	4	....[3]	5	4	55.56	.....	.....	.....	.....	.....	.....	.....	.....	170

[1] * P between .05 and .01.
† P less than .01.
[2] This ratio is within the probability range of a 2:1 ratio.
[3] Too few progeny sexed to be a reliable guide.

Negative sons of sires used in 1932

No. of each sex observed		Sex ratio	No. of each sex of malposition		Sex ratio of malposition
Males	Females		Males	Females	
.....	.....	.....	.....	.....	.....
39	54	41.58	24	42	36.36
50	55	47.62	34	35	49.28
.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....
.....	.....	.....	.....	.....	.....
39	62	38.61	25	47	34.72

of the dead embryos from these sires were examined and some of those examined were not sexed. It is to be regretted that the sex of all of the chicks that hatched from these matings was not available to be used in checking the sex ratio of the viable progeny. The primary cause and time of expression of the sex-linked lethal involved have not been determined. However, the number of embryos involved is large and lends support to the hypothesis.

Sex ratios of the non-inbred stock of this study have shown that there is normally no differential sex-ratio of dead embryos. This substantiates the findings of other workers. Jull (1931) has reported that close inbreeding of itself does not affect the sex-ratio. In the light of these facts the evidence available is convincing that a sex-linked gene is responsible for the aberrant sex ratios obtained.

The sex-ratio of the malpositions produced by a given sire is in most cases fairly close to that for all dead embryos sexed but the number of malpositions is in most cases appreciably less than the number of total sexed embryos. Obviously, then, malpositions cannot be the sole cause of the abnormal sex ratios observed but they do show a tendency toward a sex-linked ratio for some sires. In the data for two sires the sex ratio of the malpositions deviates significantly from 1:1 ratio and in several other cases the results approach a 2:1 ratio more closely than a 1:1 ratio. The differential mortality indicates that a sex-linked gene may be involved in the inheritance of malpositions.

Inheritance of Dwarfism and Interrelationships of Dwarfism, Creeper and Sticky Characters

Following the preliminary work reported by Mayhew and Upp (1932) more extensive matings were made in 1932 and 1933 to further test the mode of inheritance of dwarfism and to determine its interrelationship with the creeper and sticky characters.

In tables 25 and 26 are presented the results of matings made in 1933. Four sires and thirty-three dams, seventeen of which proved to be dwarf carriers, were involved in matings this season. The number of normal and dwarf chicks from individual matings are presented in table 25, and the normal and dwarf suspect dead-in-shell are presented in table 26.

Stickiness, described by Byerly and Jull (1930) and (1932), was also involved in one pen and the meager data available are tabulated.

All males and females were judged by appearance and were classified as carriers, non-carriers or doubtfuls prior to the breeding season. These classifications are compared to the breeding results obtained.

Totals of 73 dwarfs and 272 normal chicks were obtained from the seventeen carrier dams and 256 normal chicks and no dwarfs

TABLE 25

Inheritance of Dwarfism

Progeny ratios of individual matings of 1933

Band number		Number fertile eggs set	Number of normal chicks		Number of dwarf chicks[1]		Number of crippled chicks	Classification of breeders[4]	
Dam of chicks	Sire of chicks		Observed[3]	Expected[3]	Observed	Expected		Judged	Actual
Pen 109A									
4734	4016	60	11	12	5	4	4	0	1
4738	4016	97	24	22	5	7	3	1	1
4740	4016	72	26	23	5	8	4	0	1
4747	4016	58	10	8	1	3	0	0	1
4750	4016	2	1	..	0	..	0	0	1
Totals for carrier hens Pen 109A		289	72	66	16	22	11	..	..
4735	4016	2	2	..	0	..	0	0	??
4741	4016	51	27	..	0	..	0	0	0
4744	4016	5	3	..	0	..	0	0	??
Totals for non-producing hens Pen 109A..		58	32	all	0	none	0	.....	.....
Pen 110B									
4280	A456	17	4	6	4	2	2	0	1
4779[2]	A456	84	36	29	3	10	0	0	1
4788	A456	75	23	26	12	9	5	0	1
Totals for carrier hens Pen 110B		176	63	62	19	21	7	..	..
4778	A456	3	2	..	0	..	0	?	??
4786	A456	2	1	..	0	..	0	1	??
4787	A456	13	11	..	0	..	0	0	0
Totals for non-producing hens Pen 110B..		18	14	all	0	none	0	..	..
Pen 111A									
4844	8118	36	16	17	6	5	1	1	1
4846	8118	50	14	16	7	5	0	1	1
Totals for carrier hens Pen 111A		86	30	33	13	10	1	..	..
4795	8118	25	8	..	0	..	0	0	??
4797	8118	42	21	..	0	..	0	0	0
4798	8118	18	5	..	0	..	0	0	0
4799	8118	38	19	..	0	..	0	0	0
Totals for non-producing hens Pen 111A..		123	53	all	0	none	0	..	..

TABLE 25 (Continued)

Inheritance of Dwarfism

Progeny ratios of individual matings of 1933

Band number		Number fertile eggs set	Number of normal chicks		Number of dwarf chicks[1]		Number of crippled chicks	Classification of breeders[4]	
Dam of chicks	Sire of chicks		Observed[3]	Expected[3]	Observed	Expected		Judged	Actual
Pen 111B									
4854	3261	35	13	11	1	3	1	0	1
4862	3261	77	25	23	6	8	0	0	1
4865	3261	106	39	34	6	11	4	1	1
4867	3261	45	5	6	3	2	0	?	1
4870	3261	56	20	17	3	6	0	0	1
4871	3261	44	4	8	6	2	2	?	1
4873	3261	5	1	1	0	0	0	0	1
Totals for carrier hens Pen 111B		368	107	99	25	33	7	..	..
3705 (L)	3261	7	7	..	0	..	0	0	??
4249 (L)	3261	20	15	..	0	..	0	0	0
4858	3261	81	51	..	0	..	0	0	0
4860	3261	25	19	..	0	..	0	0	0
4866	3261	5	2	..	0	..	0	1	??
4869	3261	75	63	..	0	..	0	0	0
Totals for non-producing hens Pen 111B.		213	157	all	0	none	0	..	..
Totals for all carrier hens—1933		919	272[2]	259	73	86	26	..	..
Totals for all non-producing hens 1933 ...		412	256	all	0	none	0	..	..

[1] No chick was classified as a dwarf unless it had lived for at least two weeks and had been selected out (by appearance) as a dwarf from a random group of (circa) 30 chicks on at least two occasions at weekly intervals. Some chicks were easily identified as dwarfs at hatching time, while others were somewhat uncertain until four to six weeks of age. Some variation is tenable since classification is dependent upon external symptoms resulting from a glandular disturbance.

[2] Hen 4779 exchanged pens and produced a number of chicks sired by a Wyandotte male. Unfortunately the male was heterozygous for rose comb and all of the chicks sired by him could not be identified and eliminated. By omitting all of her chicks the totals become, observed 236:70 and expected 230:76.

[3] Probability tests of all ratios were determined by the tables of Warwick (1932) for cases in which less than 50 individuals were involved. When more than 50 individuals were concerned the X^2 test was applied.

[4] Classification of Breeders: Judged—classification prior to hatching season. Actual—classification determined by breeding results. 0—non-carrier, 1—carrier, ?—doubtful, ??—undetermined because too few progeny.

(L) Indicates purebred single comb White Leghorn hens.

TABLE 26

Dwarfism in Embryos

Dead-in-shell embryo ratios of individual matings in 1933

Dam	Sire	Number fertile eggs set	Number normal dead-in-shell (15 days or older)		Number dwarf suspect dead-in-shell (15 days or older)		Number sticky embryos	
			Observed	Expected	Observed	Expected	Observed	Expecte
Pen 109A								
4734	4016	60	25	20	2	7	0	..
4738	4016	97	30	28	13	11	0	..
4740	4016	72	15	14	4	5	0	..
4747	4016	58	6	11	9	4	12	9[1]
4750	4016	2	0	1	1	0	1	1 or 0
Totals for carrier hens Pen 109A		289	76	79	29	26	13	10
4735	4016	2	0	..	0	..	0	..
4741	4016	51	17	..	0	..	0	..
4744	4016	5	2	..	0	..	0	..
Totals for non-producing hens Pen 109A...		58	19	all	0	none	0	..
Pen 110B								
4280	A456	17	1	2	1	0	..	..
4779	A456	84	8	8	3	3	..	..
4788	A456	75	16	15	4	5	..	..
Totals for carrier hens Pen 110B		176	25	25	8	8	..	..
4778	A456	3	0	..	0	..	..	..
4786	A456	2	0	..	0	..	..	..
4787	A456	13	2	..	0	..	..	..
Totals for non-producing hens Pen 110B...		18	2	all	0	none	..	..
Pen 111A								
4844	8118	36	2	3	2	1	..	..
4846	8118	50	15	16	6	5	..	..
Totals for carrier hens Pen 111A		86	17	19	8	6	..	..
4795	8118	25	5	..	0	..	..	..
4797	8118	42	10	..	0	..	..	..
4798	8118	18	8	..	0	..	..	..
4799	8118	38	8	..	0	..	..	..
Totals for non-producing hens Pen 111A...		123	31	all	0	none	..	..

TABLE 26 (Continued)

Dwarfism in Embryos

Dead-in-shell embryo ratios of individual matings in 1933

Dam	Sire	Number fertile eggs set	Number normal dead-in-shell (15 days or older)		Number dwarf suspect dead-in-shell (15 days or older)		Number sticky embryos	
			Observed	Expected	Observed	Expected	Observed	Expected
Pen 111B								
4854	3261	35	9	8	1	2	..	..
4862	3261	77	16	16	5	5	..	..
4865	3261	106	29	25	4	8	..	..
4867	3261	45	25	19	0	6	..	..
4870	3261	56	13	11	1	3	..	..
4871	3261	44	14	13	3	4	..	..
4873	3261	5	1	2	1	0	..	..
Totals for carrier hens Pen 111B		368	107	92	15	30	..	..
3705	3261	7	0	..	0	..	..	..
4249	3261	20	5	..	0	..	..	..
4858	3261	81	9	..	0	..	..	..
4860	3261	25	1	..	0	..	..	..
4866	3261	5	2	..	0	..	..	..
4869	3261	75	8	..	0	..	..	..
Totals for non-producing hens Pen 111B...		213	25	all	0	none	..	..
Totals for carriers in 1933		919	225	214	60	71	..	..
Totals non-producing hens in 1933		412	77	all	0	none	..	..

[1] Total normals, (i. e., non-sticky) including normal chicks, dwarf chicks, normal embryos and dwarf suspect embryos make a ratio of 26 normals to 12 sticky embryos

were secured from the same males mated to 16 dams yielding negative results. The sire of a portion of the chicks from one hen is not definitely known. If the chicks of this hen be omitted, the observed ratio become 236:70 as compared to the expected, for a 3:1 ratio of 230:76. Of chicks dead-in-shell (15 days or older) examined from the carrier hens, 60 were classified as dwarf suspects as compared to 225 normals (table 26). The deficiency in the dwarf suspect class may be due to inability to recognize the dwarf embryos or it may be due to variation in time and degree of expression of the dwarf character. Seventy-seven normal dead-in-shells and no dwarfs were examined from the negative hens.

Particular mention is made of hen 4249 and her daughters. During the 1932 season this White Leghorn hen was mated to a proven dwarf carrier, a Rhode Island Red male, and produced 31 normal chicks and no dwarfs. In 1933 she was mated to her crossbred son that also proved to be a dwarf carrier and produced 15 normal chicks and no dwarfs. Eight daughters of 4249 sired by the known carrier Red male used in 1932, were included in the 1933 matings. Four of them were mated to a full brother and the other four mated to proven dwarf-producing males. Three of those mated to their brother proved to be carriers and one a non-carrier. One of those mated to other positive males proved to be a carrier, and three gave negative results. The four carrier daughters produced 80 normal and 22 dwarf chicks, while the negative daughters produced 111 normals and no dwarfs. The dwarfism character was thus transmitted by, but not expressed in, the F_1 crossbred birds and reappeared in both sexes in the F_2 generation as would be expected of a simple autosomal recessive.

Pen 109-A was a brother-sister (F_1) mating with the exception of two pullets and one of these was a half-sister to the sire. The breeders in this pen, except the two noted, were the 1932 offspring of a stickiness carrier hen obtained from the U. S. Department of Agriculture experimental farm at Beltsville and a dwarf carrier male. It was determined during the 1932 season that the two sticky hens, 6220 and 6223, carried dwarfism as well as stickiness. A sibling mating was made in 1932 in an attempt to determine the inter-relationship of dwarfs and stickiness. In view of previous results it was surmised that some pullets might carry dwarfism, others stickiness, still others both of these characters, and some pullets neither of them. Fortunately the sire heading this pen carried both stickiness and dwarfism. Unfortunately a number of the pullets could not be induced to lay in trapnests, consequently no hatching record was obtained from them and only a limited number of chicks was hatched from some others. Two hens produced sticky embryos although in only one case was the number identified of appreciable size. Hen 4747 produced 12 sticky embryos to 26 non-sticky embryos and chicks. This hen was also a

dwarf producer, her totals for the season being 10 normal chicks to 1 dwarf and 6 normal dead-in-shell to 9 dwarf suspect dead-in-shell or combined totals of 16 normals to 10 dwarfs. Three of the sisters produced dwarfs but no stickys and one produced 27 normal chicks and 17 normal dead-in-shell with no dwarfs or stickys. In no case did a hen produce sticky chicks but no dwarfs. The numbers involved are small but it appears that dwarfism and stickiness are inherited as separate characters.

During the past two years a considerable number of crippled chicks appeared in various dwarf matings. These chicks walk on their "hocks" and apparently have no use of their feet and shanks. The left leg, the right leg or both may be affected and the characteristic may be present at hatching time or may develop even after the chicks are several weeks of age. Chicks have been observed that were crippled yet were not dwarfs; but only three cases were noted this past season in which crippled was not associated with dwarfism as compared to 25 cripples that were also dwarfs. Only about one-half of the dwarf-producing dams produced any crippled chicks. No attempt is made at this time to explain the inheritance of this condition.

The phenotypic classification of the breeders prior to the hatching season, as judged by appearance, is compared to the proven classification as determined by subsequent breeding tests. The results are of interest although the numbers involved are too small to be conclusive.

All proven non-carriers, seven in all, were so judged, and of the six judged as carriers, four proved to be positive and the remaining two had too few progeny to be proven. Of the total of seventeen proven carriers, four were judged as carriers, three as doubtful and ten as non-carriers. In other words, it appears that some dwarf carriers can be detected by appearance prior to a breeding test while others cannot be so selected.

It is perhaps significant to note that while the number of dwarfs is, in general, below the expected number and ratios of individual sires and dams vary widely; yet in none of the 43 matings is the ratio outside the probability range.

All data available are summarized in table 27. The grand totals from 33 dams and 10 sires were 464 normals to 123 dwarfs, as compared to an expected ratio of 441:146. If, however, the data of hen 4779 are omitted (the case wherein some uncertainty exists) the observed ratio becomes 428:120 and expected ratio 411:137.

Totals of 388 normal chicks and no dwarfs were observed from 23 other dams mated to eight of the dwarf-producing males.

TABLE 27

Results of Individual Matings Prior to 1933

Grand Totals for All Data

Dam's number	Sire's number	Total number of chicks	Number of normal chicks		Number of dwarf chicks	
			Observed	Expected	Observed	Expected
Warren (1933b) Data from Kansas Agr. Exp. Station						
(1927)						
8448	978M	24	18	18	6	6
8388	978M	19	16	14	3	5
Total Sire	978M	43	34	32	9	11
(1928)						
8448	1037M	19	16	14	3	5
1394A	1037M	17	16	13	1	4
1404A	1037M	9	7	7	2	2
1414A	1037M	18	14	14	4	4
Total Sire	1037M	63	53	48	10	15
Carrier hens and carrier males: Data from Mayhew and Upp (1932)						
33	1	6	5	4 or 5	1	1 or 2
39	1	27	21	20	6	7
50	1	7	6	5	1	2
30-12	100	17	14	13	3	4
Totals for carrier sire matings.....		57	46	43	11	14
Same carrier hens and negative males						
33	99 and F425	22	22	..	0	..
39	98, F425, & 34	25	25	..	0	..
50		5	5	..	0	..
30-12	98	11	11	..	0	..
Total carrier hens mated to negative males		63	63	all	0	..

TABLE 27 (Contnued)

Results of Individual Matings Prior to 1933

Grand Totals for All Data

Dam's number	Sire's number	Total number of chicks	Number of normal chicks		Number of dwarf chicks	
			Observed	Expected	Observed	Expected
Data from Louisiana Agr. Exp. Station 1932						
Pen 109						
4245	A456	11	9	8	2	3
4248	A456	11	8	8	3	3
6220[1]	A456	23	21	17	2	6
6223[1]	A456	7	3	5	4	2
Total carrier hens mated to carrier males		52	41	39	11	13
Pen 109						
4246	A456	2	2	..	0	..
4247	A456	7	7	..	0	..
4249	A456	31	31	..	0	..
Leg.						
Total for negative hens Pen 109...		40	40	all	0	..
Carrier hens mated to carrier males						
Pen 102						
4621	8118	25	17	19	8	6
4630	8118	2	1	2	1	0
Total carrier hens and carrier males Pen 102		27	18	21	9	6
Same carrier hens mated to negative males						
4621	398	15	15	..	0	..
4630	398	14	14	..	0	..
Total carrier hens and negative males Pen 102		29	29	all	0	..
Summarized data for 1933—Louisiana Agr. Exp. Station						
Totals producer matings 1933.....		345	272	259	73	86
Totals non-producer matings 1933..		256	256	256	0	0
Grand total all producer matings..		587	464[2]	440	123	147
Grand total all non-producer matings		388	388	all	0	0

[1]Hens 6220 and 6223 were proven sticky carriers but produced no sticky chicks (from a total of 30) when mated to male A456, a proven dwarfism carrier. This lends further support to the contention that dwarfism and stickiness are distinct characters.

[2]If data on hen 4779 are eliminated ratios become (Observed 428:120), (Expected 411:137).

It is concluded that dwarfism is inherited as a simple autosomal recessive.

Table 28 presents detailed data showing the incidence of dwarfism by sexes, for chicks and for dead embryos. Examination of these data reveals that dwarfism occurred in the two sexes with equal frequency.

Dwarfism, chondrodystrophy, the sticky character and the creeper character are similar in that abnormal bone development is involved in all of them. In dwarf, chondrodystrophic, and creeper individuals a decided shortening of the leg bones is apparent, Landauer (1933). Chondrodstrophy affects embryos in a manner quite similar to that in which dwarfism affects chicks. These observations led to speculation as to whether or not some of these conditions might be caused by the same genes. Certain matings were made to ascertain the relationships of these characters.

Some evidence that the dwarfism, creeper and sticky characters are distinct abnormalities, is presented in table 29.

A total of seventy normal (as to dwarfism) chicks and no dwarfs produced from matings in which one parent was a creeper and the other carried dwarfism indicates that these characters are inherited independently. A sire carrying dwarfism and stickiness mated to a creeper dam produced six normal dead-in-shell embryos and chicks and no sticky embryos. This is insufficient evidence from which to draw conclusions. Totals of 36 heterozygous creepers to 34 normals were obtained from matings in which one parent was a creeper; while 10 normals, 29 heterozygous creepers, and 11 homozygous creepers were produced by creeper X creeper matings. The observed numbers are close to those expected from such matings. The apparent interrelationships of these characters may be summarized in a few statements. Chondrodystrophy, as revealed by data previously given in this report, was not especially associated with any of the above types of matings. The stickiness and dwarfism traits were likewise shown to be inherited independently and the creeper character to be distinct from dwarfism. Adequate data were not obtained to test the relationship of the stickiness and creeper traits in inheritance.

TABLE 28

Dwarf Producer Matings 1933—Sex of Embryos and Chicks

Together With Some Comparative Data from the Kansas Station

Dams	Normal Chicks			Dwarf Chicks[1]			Normal Dead-in-Shell			Dwarf Suspects Dead-in-Shell		
	Males	Females	Sex?	Males	Females	Sex?	Males	Females	Sex?	Males	Females	Sex?
Pen 109-A Sire 4016—Sire and Dam both dwarf carriers												
4734	6	3	2	1	1	3	13	11	1	1	1	0
4738	9	11	4	0	3	2	15	10	5	4	8	1
4740	10	15	1	1	3	1	6	7	2	2	2	0
4747	6	4	0	1	0	0	5	1	0	3	4	2
4750	0	0	1	0	0	0	0		0	0	1	0
Total Sire 4016	31	33	8	3	7	6	39	29	8	10	16	3
Pen 110-B Sire A456—Sire non-carrier; dam carrier. A456 himself a proven carrier last season												
4280	2	2	0	3	1	0	1	0	0	1	0	0
4779	15	19	2	2	1	0	5	3	0	2	1	0
4788	11	12	0	3	7	2	4	11	1	1	3	0
Total Sire A456	28	33	2	8	9	2	10	14	1	4	4	0
Pen 111-A Sire 8118—Himself a proven carrier last season												
4844	11	5	0	0	3	3	0	1	1	2	0	0
4846	4	9	1	1	2	4	8	7	0	2	4	0
Total Sire 8118	15	14	1	1	5	7	8	8	1	4	4	0

[1] Thirty-six dwarfs died before 4 weeks of age and thirty-seven lived to be 4 to 20 weeks of age. None attained sexual maturity; therefore dwarfism may be classed as a delayed-lethal character.

TABLE 28 (Continued)
Dwarf Producer Matings 1933—Sex of Embryos and Chicks
Together With Some Comparative Data from the Kansas Station

Dams	Normal Chicks			Dwarf Chicks[1]			Normal Dead-in-Shell			Dwarf Suspects Dead-in-Shell		
	Males	Females	Sex?	Males	Females	Sex?	Males	Females	Sex?	Males	Females	Sex?
Pen 111B Sire 3261 R. I. Red X Leghorn Crossbred—Sire carrier, dam non-carrier												
4854	5	7	1	1	0	0	4	5	0	0	1	0
4862	10	13	2	1	1	4	8	7	1	1	4	0
4865	22	14	3	6	0	0	11	17	1	3	1	0
4867	1	4	0	0	1	2	13	11	1	0	0	0
4870	7	11	2	0	2	1	7	4	2	1	0	0
4871	3	1	0	2	1	3	8	6	0	1	2	0
4873	0	0	1	0	0	0	1	0	0	0	1	0
Total Sire 3261	48	50	9	10	5	10	52	50	5	6	9	0
Totals for all known carriers—1933	122	130	20	22	26	25	109	101	15	24	33	3
(1927) Sire 978M—Data from Kansas Agr. Exp. Station, Warren (1933)												
8448	7	11	0	2	4	0	...	...	..	..	..	..
8388	6	10	0	0	1	2	...	...	..	..	..	..
Total Sire 978M	13	21	0	2	5	2	...	...	..	..	..	..
(1928) Sires 1037M												
8448	0	2	14	0	2	1	...	...	..	..	..	..
1394A	2	2	12	1	0	0	...	...	..	..	..	..
1404A	2	0	5	0	0	2	...	...	..	..	..	..
1414A	1	2	11	1	1	2	...	...	..	..	..	..
Total Sire 1037M	5	6	42	2	3	5	...	...	..	..	..	..

[1] Thirty-six dwarfs died before 4 weeks of age and thirty-seven lived to be 4 to 20 weeks of age. None attained sexual maturity; therefore dwarfism may be classed as a delayed-lethal character.

TABLE 29

Relationship of the Creeper Character to Dwarf and Sticky Characters

Number of matings	Parents		Progeny				
	Male	Female	Normals	Hetero-zygous creepers	Dwarfs	Stickys	Homo-zygous Creepers
7	Dwarf carrier	X Creeper	20	25	0	..	..
1	Creeper	X Dwarf carrier	11	8	0	..	..
1	Sticky and dwarf carrier	X Creeper	3	3	0	0	..
2	Creeper	X Creeper	10	29	..	..	11

DISCUSSION

The results have been discussed rather fully in each section as the data were presented, therefore this section is brief.

This study calls attention to the importance of breeding as a cause of variations in the embryo mortality curve.

It is worthy of mention that the incidence of abnormal embryos was greater in this material than in previously reported work. Part of this increase is due to the fact that some conditions were considered as abnormalities herein, that were not included by previous investigators. This does not account for all of the difference, however, because a direct comparison of terata of various kinds revealed more frequent occurrence of these in the present study. It is believed that the more complete examination of the dead embryos of all ages is responsible in part for the differences obtained. Breeding stock is shown to be a primary factor in the incidence of certain types of anomalies.

Chondrodystrophy did not vary seasonally in the Louisiana data as it had been reported to vary in data collected in Canada. It is an interesting speculation as to whether or not the inconsistency is due to differences in geographic location and attendant environmental differences. Curiosity is likewise aroused as to why abnormalities of the eyes occurred more frequently later in the season while no other anomaly showed this tendency. Further work is needed to verify these points.

Whether or not chondrodystrophy is inherited has not been definitely answered but further evidence is presented which supports the hypothesis that it has a complex hereditary background.

The proposed hypothesis that an impaired sense of orientation is the basic factor underlying all malpositions will be questioned. It must have additional support before final conclusions are drawn. It is believed, however, that the data lend support to this premise.

Evidence presented leads to the conclusion that a sex-linked lethal gene was present in the highly inbred single comb White Leghorn stock.

The breeding results secured indicate definitely that dwarfism is inherited as a simple autosomal recessive. A fertile field of work is available in histological and physiological studies of this character and in similar work as to the interrelationships of dwarfism, stickiness, the creeper character and chondrodystrophy.

SUMMARY AND CONCLUSIONS

1. A study has been made of embryo mortality in the domestic fowl and of the possible inheritance of the abnormalities commonly encountered. Four thousand one hundred and eighty dead embryos of known pedigree from 16,462 fertile eggs produced by 567 hens mated to 86 males were examined individually.

2. Particular consideration has been given to the age of the embryos at death, to distribution of the sexes and to incidence and descriptions of all abnormal embryos encountered; as influenced by time of hatch, by breed, by character of mating and by ancestry.

3. Mortality curves were of the same general type for the different matings included in the study but some striking minor variations were noted.

4. A minor peak of mortality at about ten days of incubation was characteristic of the White Wyandotte and Creeper matings only.

5. Particularly high mortality during the first four days of incubation was found for the stickiness, albino Wyandotte, and the creeper matings.

6. Matings involving the sticky factor showed exceptionally high 18th day mortality.

7. Closely inbred S. C. W. Leghorn matings, which were also 'high malposition' stock, gave a different type of mortality curve for the latter part of the incubation period. Mortality increased consistently from the 16th through the 21st day of incubation for this stock.

8. The major peak of mortality occured variously on the 18th, 19th, 20th or 21st day of incubation, dependent upon the character of mating under consideration.

9. The percentage of embryos that died on or after the 21st day of incubation varied for matings of different types, from 2.27 percent to 24.12 percent of the total embryo mortality.

10. It is concluded that the exact type of the mortality curve varies appreciably for different breeding stock.

11. The distribution of prenatal mortality did not show any consistent seasonal trend for eggs set on different dates.

12. From 9,110 fertile eggs set, all of the embryos that died were examined. Slightly more than 60 percent of all dead embryos, or over 15 percent of the fertile eggs set, were classified as being abnormal in some way.

13. Chondrodystrophy was noted in 6.55 percent of total dead embryos or as 1.79 percent of the total fertile eggs set.

14. The incidence of eight types of monsters was greater in this study than in previous work reviewed.

15. Eye and brain defects constituted 47.85 percent of all terata examined.

16. Chondrodystrophy, hemorrhage, malpositions and delayed hatch varied widely in occurrence for different types of matings and for different breeds.

17. Contrary to previous reports, the incidence of chondrodystrophy did not vary in this study with the season in which the eggs were incubated.

18. Abnormalities of the eyes revealed a marked seasonal trend, becoming more frequent as the season advanced. The incidence of hemorrhage decreased for later hatches, but the trend was not pronounced. Season had little affect, however, upon the occurrence of most types of abnormal embryos.

19. The sex ratio of dead embryos was not influenced by the date the eggs were set.

20. When considered by sires, the incidence of eversion of viscera varied at random, while abnormal eyes, delayed hatch, hemorrhage, and edema varied more according to sire than could be attributed to chance. No evidence of the presence of genetic ratios was apparent.

21. The incidence of total malpositions was independent of sex for all matings except the highly inbred stock. In this case 63.27 percent of all malpositions were females. However, on a basis of percentage of each sex that died the incidence of malpositions was similar for each of the sexes in the inbred matings also.

22. Certain sires produced a positive excess of dead female embryos and others produced sex ratios that were probably abnormal.

23. Chondrodystrophy varied too greatly in fifty-three individual matings tested to be accounted for by chance variation. Limited breeding data suggest that chondrodystrophy may be inherited. It is concluded that, if inherited, chondrodystrophy is recessive in character with at least three pairs of factors involved.

24. Malposition 1, head between legs, malposition 4, rotated in shell, and malposition 5, head not under wing, occurred at approximately the same rate in the non-inbred stock of this study. In the highly inbred stock, in which the incidence of malpositions was much higher, malposition 5 appeared much more frequently than did any other position.

25. It was observed that a given dam or a given sire often produced several different types of malpositions. The regularity with which this occurred suggests the hypothesis that no innate differences

underlie specific positions, but rather that an impaired sense of orientation is perhaps the primary consideration.

26. Incidence of malpositions was much higher in the inbred stock and it increased markedly in the inbred matings of 1933 as compared to those of 1932. These facts are considered as evidence that breeding is an important factor in the occurrence of malpositions.

27. Abnormal sex ratios of dead embryos were produced by certain highly inbred S. C. W. Leghorn males and by some of their sons. It is concluded that a sex-linked lethal gene was responsible for the aberrant sex ratios observed.

28. An increased incidence of female malpositions was noted in the matings which produced the aberrant sex ratios.

29. Breeding tests concerning the inheritance of the delayed lethal character, dwarfism, involving 32 dams and 10 sires, produced 428 normals and 120 dwarfs as compared to an expected ratio of 411:-137. It is concluded that dwarfism is inherited as a simple autosomal recessive.

30. A condition in chicks designated as crippled was associated with dwarfism in 25 cases and appeared independently of dwarfism in 3 cases. The mode of inheritance of this condition has not been determined.

31. As shown by subsequent breeding tests, some carriers of dwarfism are recognizable by appearance while others are not.

32. Breeding results indicate that (a) dwarfism and stickiness are controlled by separate genes, (b) creeper and dwarfism are distinct traits and (c) chondrodystrophy is not associated in inheritance with any of these characters.

33. Adequate data were not obtained to test the relationship of the stickiness and creeper traits in inheritance.

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